ARCHIVES of OPHTHALMOLOGY

VOLUME 4 (old series Volume 61) NOVEMBER, 1930 NUMBER 5

# THE TREATMENT OF DETACHED RETINA BY SEARING THE RETINAL TEARS*

PROF. DR. J. GONIN
LAUSANNE, SWITZERLAND

As I was entrusted by my teacher, Prof. Marc Dufour, with the preparation of the chapter on the diseases of the retina for the French "Encyclopedia of Ophthalmology," I felt obliged to recognize the truth of the explanation given by Leber of Heidelberg for the sudden forms of retinal detachment. Leber, emphasizing the fact that an exudation from the choroid should necessarily increase the intra-ocular tension (while it is known that hypotomy is rather more frequent in eyes with detachment), admitted that the source of the subretinal fluid was the liquid part of the vitreous which passed into the subretinal space. This explanation was based on the frequent presence of a crescentic or horseshoe-shaped tear in the retinal tissue, the flap always projecting into the vitreous body with its apex toward the papilla and its base to the periphery, these peculiarities showing that the tear was due to traction on the retinal tissue from behind forward and inward.

The anatomic examination of some relatively recent cases of detachment enabled me in 1904 to complete with greater precision Lebers' theory, and in a report or a symposium which the French Ophthalmological Society asked me to write in 1920, I gave the following conclusions on the basis of a greater anatomic and clinical material:

(*a*) In most, if not in all cases of so-called "spontaneous" or "idiopathic" detachment, one or several tears may be seen in the retina.

(*b*) The production of the tears is due to the vitreous humor dragging on the retina at the site of previous adhesions between both tissues, those adhesions having been formed near disseminated foci of chorioretinitis, which are frequent in myopic and senile eyes.

(*c*) The presence of a hole, allowing the fluid of the vitreous to pass behind the retina and maintaining thus the subretinal collection of liquid, explains the ophthalmoscopic appearance of the "spontaneous" detachment much better than the hypothesis of an exudation from the choroid. In the forms caused by such an exudation (detachment of pregnancy, in Coat's disease or associated with tumors or acute inflammations, etc.), the clinical course is also different.

* Submitted for publication, July 15, 1930.

* Part of an address read before the Oxford Congress, July 12, 1930.

As the retinal tear was the determining cause of the "spontaneous" detachment and prevented it from healing, it was obvious that it was impossible to secure permanent cure by any of the therapeutic methods hitherto in use, the danger of a recurrence lasting as long as the hole in the retina remains open. Particularly the many treatments the purpose of which is to give exit to the subretinal fluid or to promote its resorption, such as scleral punctures or draining, diaphoresis, subconjunctival injections, etc., were condemned as useless, as they did not hinder the continual passing of liquid through the rent in the retina. This explains the habitual failures of those treatments and the general skepticism that is met with among most ophthalmologists, many of whom think that it is not even worth while to make an attempt at cure. Professor Haab of Zurich, has lately written me that when he was in America he asked one of the most prominent oculists what treatment he generally used for patients with retinal detachment. "I send them to the most disagreeable of my colleagues," was the answer. That this special method did not prove to be sufficient as a rule is shown by the well known statistics of Dr. Vail, of Cincinnati, who in 281 replies from oculists in the United States whom he asked for information found that 250 of them never cured a single patient; of the 31 who obtained cures, 25 obtained a single cure each, so that permanent success occurred in hardly 1 in every 1,000 cases. Dr. Vail's conclusion, therefore, was that any vigorous treatment was to be refrained from until a "cure" should be found that really cures.

The British ophthalmologist who, I think, is at present the best informed on retinal detachment, Sir William Lister, has come to the same pessimistic conclusion as our American colleague, judging from the address he gave in 1927 before the British Medical Association.[1] Holes in the retina, he said, are often seen, and they probably occur more often than they are found even by those who look for them, but it would seem that in such cases treatment is practically valueless and "should not be urged except as a last clutch at a straw of hope."

I was of the same opinion as Sir William Lister, until I thought of the possibility of closing up the tear and checking at the same time the dragging force of the vitreous on the retinal flap. I then succeeded in doing so by means of a thermopuncture through the sclera, first in some cases of rather old detachment, and obtained a complete reposition of the retina, although, of course, its visual function was not restored. With greater courage, I then tried the same operation in more recent cases and even in cases in which the patient had vision in one eye, and obtained the astonishing result that not only the progress of the detachment was stopped and the retina reapplied near the cauterization, but that even in widely spread detachments, the whole

1 Lister, William T.: Brit. M. J. **2**:1127 (Dec. 17) 1927.

fundus of the eye had recovered a normal appearance with no further trace of folds or of grayish discoloration. In a certain number of cases, central vision and peripheral field were restored to normal so that the formerly detached eye remained the better one, being used even for watchmaker's work.

These unexpected successes were first referred to before the Swiss oculists at Bâle in 1919, with, of course, great reserve, then in some Swiss or French reviews, such as *Annales d'oculistique,* 1921, and *Revue générale d'ophthalmologie,* 1923, later, with a short description of some typical cases presented before the French, Swiss and German Societies in Brussels, Zurich and Heidelberg in 1925 and 1926. In 1927, there was shown at Bern several patients, the complete disappearance of whose detachments lasted for several months, and in the following year, at Lucerne, my colleagues, Siegrist of Bern and Vogt of Zurich, referred to their own confirmatory experiences; this was the first occasion on which credit was given to me by ophthalmologists other than my own fellow-workers and assistants. Later descriptions of cases or of the method were published in Italian, French or German in the reports of the ophthalmologic meetings held at Palermo, Paris and Heidelberg in 1928, and a more detailed account was published in the *Annales d'oculistique* (May, 1930).

My actual experience on about 250 patients and in more than 300 operations can be summed up as follows:

1. In more than 95 per cent of the cases, whenever ophthalmoscopic examination is possible, one or several holes may be detected in the retina if looked for with sufficient care.

2. In about 10 per cent of these cases, the hole is not in the retinal tissue, but consists of a rupture or tearing away from its insertion at the ora serrata ("desinsertion of the retina").

3. In all recent cases, when the hole or tear has been closed, cure is immediate, complete and permanent.

4. In older cases (several weeks or months), closing the tear stops the detachment and may produce a more or less complete reposition of the retina, but restoration of the vision generally remains incomplete.

5. If the detachment relapses, it is found that the tear has not been completely closed or that there was another tear which had not been previously seen.

6. A recurrence of detachment in a different region of the eye is due to formation of a new hole in the retina.

It may appear curious that all my communications on that matter, numbering more than twenty, remained unknown to the English-speaking ophthalmologic world until the International Congress in Holland, no mention of them even being made either in an article

in the *Lancet* [2] or in an article by Stallard [3] in the *British Journal of Ophthalmology*. Only following a recent visit to me at Lausanne by five members of the staff of the great eye hospitals in London were my ideas set forth in two papers published by Juler [4] and by Ormond.[5] The former described accurately the preliminary procedures [6] and the operation of thermopuncture; the latter dealt adequately with the anatomic side of the question.[7]

As both of these papers gave a sufficient account of my ideas, I will not repeat the particulars but I wish to emphasize the great importance of being acquainted with the conditions causing a detachment rather than with the routine procedure of the operation itself. I fancy the latter could be done in a different manner than mine, but the pathogenesis of the detachment is not a matter of personal preference, for truth must be accepted such as it is. Some of the visitors seemed to have an insufficient knowledge of the facts underlying the method, so that I could not imagine what benefit they might derive from their visit. It is surely of little value to witness an operation if one is unable to understand not only the how but also the why of the procedure. Before inquiring about the technic of, and performing the thermopuncture, it is necessary to be well aware of anatomic and clinical reasons.[8]

In the *American Journal of Ophthalmology*,[9] Gradle of Chicago made some witty remarks about the pilgrimage to Lausanne, which he calls the Mecca of ophthalmologists, and about the green turban of the pilgrims. Nevertheless, his account of the pathogenesis as I describe

---

2. Operation for Detachment of the Retina, Lancet **2**:1202 (Dec. 7) 1929.

3. Stallard, H. B.: Brit. J. Ophth. **14**:1 (Jan.) 1930.

4. Juler, Frank: Brit. J. Ophth. **14**:73 (Feb.) 1930.

5. Ormond, A. W.: Brit. M. J. **1**:940 (May 24) 1930.

6. The whole procedure of locating the tear and preparing for the operation, as seen by Dr. de Jaeger of Bruges at Lausanne, has been again described by his partner, Dr. Rubbrecht, whose paper is summarized with full particulars and sketches, by J. B. Lawford in the British Journal of Ophthalmology (**14**:359 [July] 1930). The only difference in Dr. Rubbrecht's method is that he makes a long scleral wound for the cauterization.

7. On a single point of importance, Ormond seems not to have realized my own conception of the changes in the vitreous. He stated that "liquefaction of the vitreous takes place." I do not think it does. I noticed only a separation of the vitreous fluid from its fibrillar mass, the latter being penetrated with epithelial cells and keeping thus a retractile strength sufficient to drag on the retina. I generally compare the retracted vitreous with a squeezed lemon, the cellular part of which has been not liquified but simply deprived of its juice.

8. Gonin, J.: Ann. d'ocul. **132**:30, 1904; Encyclopédie française d'ophtalmologie, Paris, O. Doin, 1906, vol. 6; Ann. d'ocul. **156**:281, 1919; Bull et mém. Soc. franç. d'opht. **23**:1, 1920; Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **45**:114, 1921.

9. Gradle, Harry S.: Am. J. Ophth. **13**:304 (April) 1930.

it is on some points erroneous; considering the matter dispassionately, as he says, and "away from the magnetic personality of Gonin," he appears not to have understood the important part played by the vitreous in the formation of the tears and in the hindrances to their closing.

He remains unable to explain the horseshoe shape of the tears or the reason why in one case the cauterization on the edge of the tear is successful and in another case unsuccessful. Just the instance he alludes to is by no means convincing, for the patient whom he directed to me for treatment had on his arrival an iridocyclitis with severe hypotomy, so that I at once made a bad prognosis, and declared to the patient that considering the long journey he had made and his wish to be treated, I would make an attempt to cure, but that I had little confidence in the possibility of checking the general retraction of the vitreous. If Mr. Gradle had been the happy possessor of a green turban, I think he would perhaps have realized the difference between such a case and another in a better anatomic condition, but there is no need of any pilgrimage for it; it should be sufficient to be acquainted with the content of the aforementioned papers on the subject.

## SUMMARY

In summarizing my experiences to date, I should say that definite cure may be obtained in about 60 per cent of recent cases, this percentage diminishing with the age of the detachment. Not only is this proportion of cures an improvement in comparison with the former rarity, but the length of the treatment is far shorter than the many weeks or months of absolute rest in bed with both eyes bandaged, which was up to this time the most usual torment for the patients.

In the *Lancet* of December, 1929, it was said that: "There are few ophthalmic surgeons who cannot count on the fingers of one hand the cases of cure of detached retina within their own personal experience."

I should be very unhappy and quite unable to operate if I had on my two hands as many fingers as I can count patients who for the last three years have been completely and lastingly cured as rapidly as if they had simply been operated on for cataract. I trust these facts will offer my honored colleagues, Vail and Lister, more than "a straw of hope" for the benefit of their patients, and that even "away from the magnetic personality" of the writer, all oculists will find a new courage for the treatment of detached retinas, so that they may soon acquire with their own experience many successful cures.

# THE ACUITY OF BINOCULAR DEPTH PERCEPTION IN HEMIANOPIA *

E. V. L. BROWN, M.D.
AND
P. C. KRONFELD, M.D.
CHICAGO

The analysis of the various phenomena of binocular depth perception has shown that all the contributing factors can be summarized under two headings: (1) the peripheral process and (2) the central process.

In the peripheral process the most important factors are the central and peripheral visual acuity in each eye, the symmetry of vision in the two eyes and the accuracy of the action of the motor apparatus. All of these points have been studied carefully in their relations to the fineness of binocular depth perception, with the result that it is generally known to what extent anisometropia or higher degrees of heterophoria impair the acuity of depth perception.

On the other hand, it was recognized a long time ago that a complex of cerebral processes was necessary to transpose horizontal disparity and all the factors of monocular depth perception into the subjective impression of relief in the visual space. The term "central factor" was introduced for this part of the perception of perspective. Unfortunately, there is no agreement as to the definition of this central factor. Hofmann [1] defined it as the "unconscious attempt of correlating the two single images with the one object that we actually see." A similar description of the central process was given by Verhoeff [2] in his theory of binocular perspective in 1902. Verhoeff assumed:

> A separate cortical system for each eye, and connecting the two systems and bringing them into relation with one another a third system, composed of a more or less complicated arrangement of neurones upon which the binocular perception of relief is dependent. This intermediate system may thus be regarded as the cerebral representative of the hypothetical cyclopean eye of Hering and others, and may be spoken of as the cyclopean center, and with it all ideas concerned with binocular relief would be associated. . . . It is not assumed that the cyclopean center is necessarily anatomically intermediate in position with relation

* Submitted for publication, June 16, 1930.

* From the Eye Clinic of the University of Chicago.

* Read at a Meeting of the American Ophthalmological Society, Hot Springs, Va., June 2, 1930.

1. Hofmann, F. B.: Raumsinn des Auges, Ergebn. d. Physiol. **15**:238, 1915.

2. Verhoeff, F. H.: A Theory of Binocular Perspective, Am. J. Physiol. Optics **6**:416, 1925.

to the monocular systems. Knowledge regarding the anatomical relations of those portions of the brain concerned in vision is still too slight for an attempt to be made definitely to locate the cyclopean center.

Since this publication of Verhoeff, Bárány [3] and Kleist [4] have published theories concerning the parts of the visual cortex where the monocular and the cyclopean image is formed. Kleist thought that the two layers of ganglion cells that accompany the stria of Gennari represented the two areas where the monocular images are formed. These two layers (usually called IV a and IV c) are not equally thick. Therefore, it was thought that IV a, or the more superficial one (lamina granularis interna superficialis), represented the homolateral eye, while the deeper one, IV c, belonged to the contralateral eye. The big star cells which Cajal found in the layer between the two mentioned may have the function of combining the two monocular impressions into one binocular impression. They may represent what Verhoeff called the cyclopean center.

Up to this point the process of binocular depth perception is based on the fusion of two disparate images. This process, according to Best,[5] should be separated from "the central factor," by which he means the influence of the conception of shape, space and size in general added to that of horizontal disparity, which leads to the final complex sensation of depth.

From this brief review it can be seen that some authors do not consider the processes that take place in the calcarine fissure as parts of the central process, while others do. This discrepancy, however, does not interfere with the use of the term "central factor." Lesions of the brain which lead to disturbances of depth perception have been described many times. Most of these cases were injuries sustained during the World War, and from careful studies of these cases it almost seems possible to localize these lesions.

Poetzl [6] thought that the geometric-optic agnosia could be localized in the dorsal and median parts of convexity of the cuneus. If there is a definite area in which the mentioned central processes of depth perception take place, then it must lie in the neighborhood of that for geometric-optic agnosia.

When we started to work on the depth perception of certain patients, the situation was such that anomalies of the peripheral receiving apparatus and lesions of the terminal station in the brain had been exten-

3. Bárány, R.: J. f. Psychol. u. Neurol. **31**:5, 1925.

4. Kleist, K.: Klin. Wchnschr. **5**:3, 1926.

5. Best, F.: Ergebnisse der Kriegjahre für die Kenntnis der Sehbahn und Sehzentren, Zentralbl. f. d. ges. Ophth. **3**:241, 1920.

6. Poetzl, O.: Ueber die raeumliche Anordnung der Zentren der Sehsphaere des menschlichen Gehirns, Wien. klin. Wchnschr. **31**:745, 1918.

sively studied with respect to their possible relation to the acuity of depth perception. The intermediate processes, the conduction of impulses through the visual pathway and their first reception in the calcarine fissure, had been almost neglected. This was astonishing in view of the fact that Heine [7] had based his theory of bicerebral representation of the macula on the presence or absence of binocular depth perception in cases of hemianopia.

Heine's theory was as follows: Binocular vision is based on the blending of the two images of the two eyes in the cortical center. If the two images fall on corresponding points, the process of blending takes place very easily, because the fibers which conduct the stimuli from corresponding points to the cortex end in nearby points of one

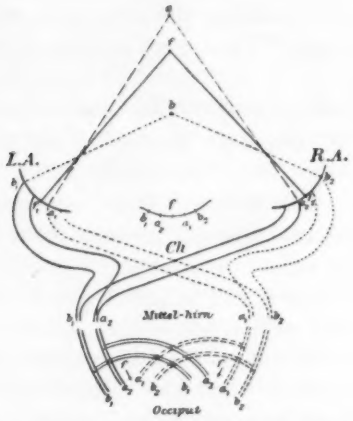



Fig. 1.—The visual pathway (after Heine).

occipital lobe. Binocular perception of depth is based on the formation of double images which have some horizontal disparation. If one considers only the perception of differences of depth in the center of the visual fields, these double images fall on heteronymous parts of the retinas (bitemporal if the second object is nearer than the one fixed, $b_1$, $b_2$, figure 1; binasal, if the former is farther away than the latter, $a_1$, $a_2$); one of them is conducted to the left hemisphere, the other to the right. These double images, however, lead to the perception of depth, and therefore it is necessary to assume association fibers between the two hemispheres. These fibers, which under normal circumstances permit the coordinated action of the two cortical foveae, become still more important if one occipital lobe is destroyed. Then they form a connection between the optic radiation of the destroyed lobe and the

7. Heine, L.: Sehschaerfe und Tiefenwahrnehmung, Arch. f. Ophth. **51**:146, 1900.

visual cortex of the contralateral side. According to Heine's theory, these fibers conduct only those impulses which fall on the retinal foveae and their immediate neighborhood. The size of this part of the visual field, which is represented in both occipital lobes, was determined by Heine, who took the acuity of binocular depth perception as the criterion, and was found to have a diameter of approximately 8 degrees.

In cases of cerebral lesions which affect the visual radiation distal to the point where the association fibers branch off and cross over to the other occipital lobe, Heine postulates a complete hemianopia with the separating line going straight through the fixation point (fig. 2). These patients should have no binocular perception of depth in the center of their visual field, or as Heine called it no central perception of relief,

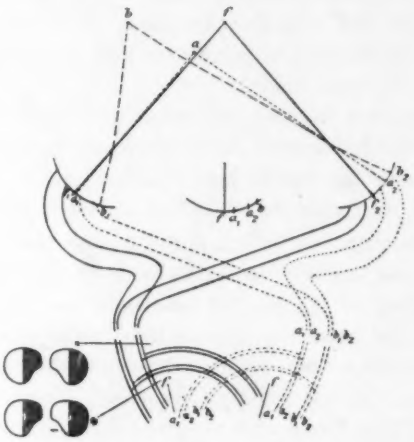


Fig. 2.—The visual pathway (after Heine).

because only one of the heteronymous single images is received by the visual cortex. Cases of lesions of the occipital lobe above the point where the association fibers branch off lead to sparing of the macula and no disturbance of central perception of depth.

This theory of Heine was supported by anatomic observations (Déjerine [8] and Pfeiffer [9]), and it is known now that these association fibers actually exist. Nevertheless, we could not find any work on the other part of Heine's theory, namely, the acuity of depth perception in cases of hemianopia. In spite of the extensive studies that have been made on cases of injuries, sustained during the World War, by

8. Déjerine, Y.: Sémiologie des affections du système nerveux, Paris, Masson & Cie, 1900, p. 797.

9. Pfeiffer, R. A.: Myelogenetisch-anatomische Untersuchungen über den zentralen Abschnitt der Sehleitung, Berlin, Julius Springer, 1925.

ophthalmologists and psychologists, we were not able to find any material on depth perception in cases of hemianopia in which all the other factors, namely, the peripheral apparatus and the central process, were normal.

### SELECTION OF CASES

In patients with bitemporal hemianopia the perception of depth had to be tested in the very center of the visual field, because in the periphery of the field there was only monocular vision. In none of our cases could any amount of binocular central depth perception be demonstrated. That may have been due to the fact that in none of them were the defects symmetrical and regular enough. Therefore, we wish to defer our decision concerning the depth perception in bitemporal hemianopia and to confine ourselves to cases of homonymous hemianopia without sparing of the macula. In cases in which the macula was spared, depth was perceived as accurately and as promptly as in normal cases with complete visual fields.

Altogether we have studied six cases of homonymous hemianopia. In four of them the lesion was definitely localized above the geniculate body. In two cases the lesion was localized in or below the geniculate body. In all six cases the observations were practically the same, so that we shall confine our descriptions to one case.

As stated before, these were cases without sparing of the macula. The test for sparing of the macula was done on the stereocampimeter of Lloyd. In all six cases the approaching small white target was not noticed until about one half of it had passed the border of the little circle which is used as the point of fixation. From the result of this test we concluded that there was no gross sparing; we then tested our cases on a Bjerrum screen at a distance of 8 meters. As the fixation object we used a fine white cross drawn on a dull black cardboard. Between the patient and the fixation point we put several black frames of decreasing size which we have found helpful in making fixation easier and steadier. The patient was asked to keep his eye on the center of the cross which also meant keeping his eye in the center of the round frames. We were aware of the fact that even if the patient did his very best, his "fixation" actually consisted of the oscillations which were described by MacAllister,[10] Koch, Dodge, Marx, Trendelenburg and others, and measured by Marx and Trendelenbung [11] who found them not higher than 10 arc minutes, while other authors found them as high as 40 minutes. It is easily seen that defects of the visual

10. MacAllister, C. N.: The Fixation of Points in the Visual Field, Psychol. Rev. (Monog. Suppl.) **7**:17, 1905.

11. Marx, E., and Trendelenburg, W.: Ueber die Genauigkeit der Einstellung des Auges beim Fixieren, Ztschr. f. Sinnesphysiol. **45**:87, 1911.

field near the fixation point cannot be taken with more accuracy than the accuracy with which the eyes are kept in a certain position. Therefore, we decided that sparings which did not amount to more than 15 minutes were probably not due to the presence of visual field on the side of the hemianopia, but to the physiologic unsteadiness of the eyes of the patient. We would like to call this phenomenon a pseudosparing. After about twenty-five determinations in each of our cases, the average extension beyond the fixation point was found to be 6 arc minutes.

## METHODS OF EXAMINATION

For the determination of the acuity of binocular depth perception we used an instrument which was based on the old "Staebchenversuch" of Hering. Similar

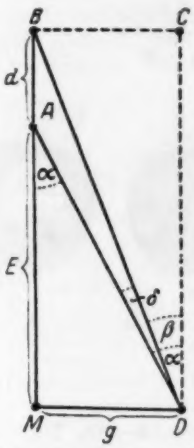



Fig. 3.—*D* represents the right eye; *MD*, one half of the interpupillary distance. The distance *AB* is the minimum difference in depth perceived. δ is one half of the binocular parallactic angle. (After Hofmann.)

instruments have been in general use for a long time. Extensive studies were made with an apparatus of this type by Harvey J. Howard and were reported at a meeting of the American Ophthalmological Society in 1919.[12] The instrument that we used was very much like Dr. Howard's. The patient was asked to decide the difference in depth between two rods shown him. The rods had a diameter of 1 cm., were painted a dead black and were shown against a white background. The patient's head was fixed by means of a chin rest and a head rest.

The acuity of binocular depth perception is usually expressed as the smallest amount of horizontal disparity which is perceived or recognized in 75 per cent of all of the readings taken. Usually the horizontal disparity is given by the binocular parallactic angle or 2 delta in figure 3. In some of his cases Howard found this angle as low as 1.8 seconds, and he recommended an angle of 8 seconds as the

12. Howard, H. J.: A Test for the Judgment of Distance, Am. J. Ophth. **2**:656, 1919.

minimum of acuity which a pilot should possess, a value which has been almost generally accepted. The binocular parallactic angle depends on various factors which must always be kept in mind if studies of the binocular fineness of depth perception are carried out. The main factors are:

1. The contrast between the rods and the background.
2. The lateral distance between the rods (approximately 1 degree in Howard's studies).
3. The time of exposure.
4. The interpupillary distance.
5. The distance between the observer and the rods.

Factors 1 and 3 were kept constant in our experiments. The time of exposure was regulated by means of a shutter which was opened for one second at a time. During this one second the patient had to decide which of the two rods seemed nearer to him.

Factors 2 and 5 were changed in our experiments. Most of our patients were tested at two distances (3.40 and 7.61 meters) from the rods. The lateral distance between the rods was changed also. Usually three series of tests were made, one with 1 degree, one with 20 and one with 10 minutes' lateral distance. In

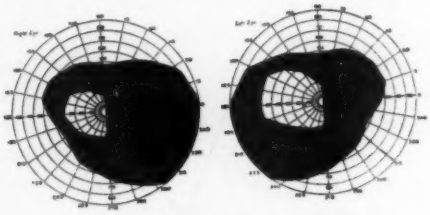


Fig. 4.—Visual fields of S. F.

each respective position of the two rods (i. e., certain anteroposterior distance and certain lateral distance) twenty readings were taken; in ten of these readings the right, and in the other ten readings the left rod was nearer to the patient. These positions were changed in an irregular sequence. The patient was told that one of the two rods would always be nearer to his eyes.

As already mentioned, the observations were practically the same in all of our six patients. We therefore confine ourselves to the description of one case which might be called a standard case in its response to the test with the two rods.

## REPORT OF CASE

*History.*—Mrs. S. F., aged 55, was referred to our clinic by Dr. Peter Bassoe, Chicago, with the diagnosis of dementia paralytica. The husband of the patient had died of dementia paralytica several years previously. The patient had never had antisyphilitic treatment. About a year previously to admission to our clinic she had "a nervous breakdown." Dr. Bassoe made the diagnosis of dementia paralytica and ordered a combined antisyphilitic and fever therapy. The patient recovered splendidly, so that for the past six months she has felt well. She does housework, has no difficulties in getting around the home, rides on the street car without difficulty and rarely bumps into things because she does not see them until she bumps into them.

*Observations.*—The patient was well nourished. The blood pressure was 155 systolic and 100 diastolic. Medical examination revealed no gross pathologic changes. Examination of the eye revealed absolute fixation of the pupils. There was a moderate degree of primary atrophy of both optic nerves, equally marked in all of the four quadrants of the disks. Moderate retinal arteriosclerosis was present. The vision of each eye was 1; under homatropine the refraction was found to be plus 1 diopter in each eye. With a reading glass the patient read the 1.5 D Snellen test type very slowly, occasionally omitting a few letters or a whole syllable at the end of a word. The visual fields showed right-sided homonymous, symmetrical and congruous hemianopia, with moderate concentric contraction of the left halves of each field (fig. 4); sparing of the macula, 14 arc minutes. In view of the optic atrophy and the sparing of the macula of only 14 minutes, we felt that we were dealing with a lesion of the optic tract. No gross disturbance of orientation was present. The subjective median plane was found to

TABLE 1.—*Right-Sided Hemianopia with Symmetrical Horopter*

| Date | Lateral Distance Between Rods in Degrees | Depth Difference Between Rods in Millimeters | Incorrect Answers (Out of 20) | |
|---|---|---|---|---|
| | | | When Right Rod Nearer | When Left Rod Nearer |
| 3/15 | 27 minutes | 30 | 3 | 1 |
| | 27 minutes | 20 | 6 | 1 |
| | 27 minutes | 15 | 4 | 1 |
| 3/20 | 1 degree | 40 | 0 | 0 |
| | 1 degree | 20 | 10 | 0 |
| 3/22 | 27 minutes | 40 | 2 | 0 |
| 3/25 | 10 minutes | 20 | 3 | 0 |
| | 1 degree | 20 | 8 | 0 |
| | 10 minutes | 20 | 3 | 0 |

be shifted slightly toward the right side. The acuity of binocular depth perception was tested several times. The results are given in table 1.

## COMMENT

The acuity of binocular depth perception that was found in our cases is surprisingly good. It amounts to a binocular parallactic angle of less than 5.5 seconds. Yet, the perception of depth could not be called entirely normal. From table 1 it can be seen that the patient always gave a certain number of incorrect readings. These readings were not equally distributed between the two possible positions of the rods, namely, the right one being nearer or the left one being nearer to the patient. In our patient most of the incorrect answers were given when the right rod was nearer to her. The frequency of this phenomenon seemed to depend on two factors, namely, the distance between the patient and apparatus, and the distance between the two rods. Under certain circumstances, all of the judgments were wrong when the right rod was the nearer one (table 2).

Before we go into the interpretation of this phenomenon, we wish to say a few words about the manner in which a normal person is most likely to behave if asked about the difference in depth between two rods. The rods are exactly alike, neither one being more attractive than the other. Therefore, a normal person probably fixes on one rod after the other. By fixing on the left one first, the right rod is seen in disparate images on the left halves of the retinas very slightly to the left of the foveae. A certain sensation of depth is achieved in this position; then the person probably fixes on the right rod, and the left one is seen in disparate images which fall on the right halves of the retinas slightly to the right of the foveae. The final impression of depth is the result of the comparison of the sensations obtained by this alternating fixation. The impression of depth depends on the relation between the actual position of the two rods and the horopter which is the locus for all the subjectively equidistant points. The horopter in the distances which we

TABLE 2.—*Judgments Given by Patient in Answer to Questions*

| | Question: Which Stripe is Nearer to You? | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Actual position of stripes | L | L | R | R | R | R | R | L | L | R | L | L | R | L | L | R | R | L | L | R |
| Answers: | | | | | | | | | | | | | | | | | | | | |
| Correct | .. | .. | + | + | + | + | + | .. | .. | + | .. | .. | + | .. | .. | + | + | .. | .. | + |
| Incorrect | + | + | .. | .. | .. | .. | .. | + | + | .. | + | + | .. | + | + | .. | .. | + | + | .. |
| | Question: Which Stripe is Farther Away from You? | | | | | | | | | | | | | | | | | | | |
| Actual position of stripes | R | L | R | R | L | L | R | L | L | R | L | L | R | R | R | L | R | L | L | R |
| Answers: | | | | | | | | | | | | | | | | | | | | |
| Correct | .. | + | .. | .. | + | + | .. | + | + | .. | + | + | .. | .. | .. | + | .. | + | + | .. |
| Incorrect | + | .. | + | + | .. | .. | + | .. | .. | + | .. | .. | + | + | + | .. | + | .. | .. | + |

used in our experiments (from 3 to 7 meters) represents in general a circle with a very small curvature, the convexity of which is toward the observer (fig. 5). If we carried out our experiments by using three rods and determined the position in which the three rods would appear equidistant, the result would be that the middle rod would be put a little nearer to the eyes of the observer than the two rods on the sides. By experimenting with two rods, this phenomenon does not occur if the horopter is symmetrical in the person tested, because he bases his final decision on a comparison of the left half with the right half of the horopter.

The hemianopic person behaves differently. He has only one half of his visual fields; therefore only one half of his horopter is at his disposal. He bases all of his judgments on the sensation obtained in one position of his eyes, namely, in a case of right-sided hemianopia, by fixing his eyes on the right rod while the left rod is forming disparate images in the two right retinal halves slightly to the right of the foveae. The hemianopic person cannot compare the result obtained in this position with that of any other position. Therefore, the hemi-

anopic person is bound to show the curvature of the horopter much more markedly than the normal person.

In our case, in which the patient had right-sided hemianopia, only the left half of the horopter remained, which meant that near the fixation point the left rod had to be put slightly farther away than the right one in order to appear equidistant to the patient. This was the only symptom which differentiated these six hemianopic patients from a person with normal complete visual fields.

In order to prove that the phenomenon that we observed in our patients actually represented the configuration of the horopter, we varied the distance between the two rods. As the horopter is a circle of a certain curvature, it shows this curvature much less if the two rods are placed on it with a shorter distance between them. Table 1

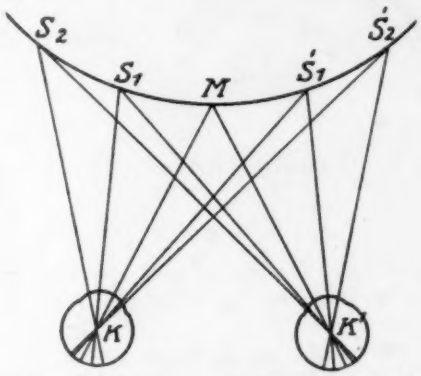



Fig. 5.—The horopter (after Hering-Hillebrand).

shows this in the case reported. The closer the rods were put together, the less frequent became the incorrect statements of the patient.

The phenomenon that we have found in hemianopia can also be seen in persons with normal visual fields. It was mentioned in the old literature, and recently Howard drew attention to it. He found "in 23 cases out of the 106 subjects a tendency to project one image nearer than the other. Although some of these cases had muscle imbalance, others visual asymmetry and still others had both these abnormal conditions, the phenomenon cannot be explained satisfactorily."

Howard did not examine the refraction of his cases if the vision was found normal. Therefore, it is likely that many of his twenty-three cases actually had visual asymmetry although the unaided vision was found equal. The experiments of Erggelet [13] on artificial aniso-

13. Erggelet, H.: Versuche zur beidaeugigen Tiefenwahrnehmung bei hoeherer Ungleichsichtigkeit, Klin. Monatsbl. f. Augenh. **66**:685, 1921.

metropia and the studies of Fischer [14] on asymmetries of the visual organ showed that the horopter is asymmetrical in these cases; for instance, in Erggelet's experiment the horopter as a whole was tilted toward one side. Fischer found on himself that the apex of his horopter for white was not located in his point of fixation, but in a slightly eccentric point (fig. 6). In all these cases of visual asymmetry which, in our experience, are quite common, asymmetries of the horopter must be expected. This accounts for the tendency of projecting two equidistant objects in different planes.

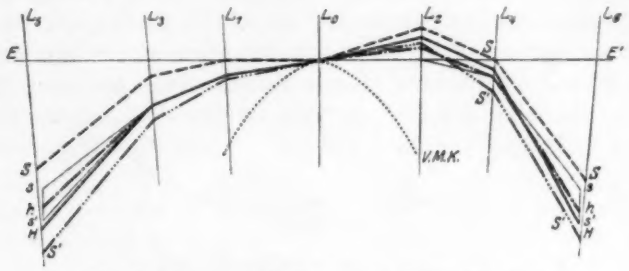



Fig. 6.—Asymmetrical horopter (after Fischer).

TABLE 3.—*Left-Sided Hemianopia with Asymmetrical Horopter*

| Date | Lateral Distance Between Rods in Degrees | Depth Difference Between Rods in Millimeters | Incorrect Answers (Out of 20) | |
|---|---|---|---|---|
| | | | When Right Rod Nearer | When Left Rod Nearer |
| 3/21 | 27 minutes | 80 | 0 | 0 |
| | 27 minutes | 40 | 6 | 0 |
| | 27 minutes | 20 | 10 | 0 |
| 3/23 | 27 minutes | 60 | 4 | 0 |
| | 27 minutes | 40 | 5 | 0 |

In one of our patients with hemianopia we found a marked visual asymmetry, and accordingly an asymmetrical horopter. It was a case of left-sided hemianopia with the apex of the horopter about 10 degrees to the right of the fixation point. Consequently, the left rod had to be put farther away than the right one in order to appear equidistant (table 3).

Although we felt that the described phenomenon in hemianopia was fully explained by the fact that there was only one half of the horopter left, we thought of another possibility. *A priori,* it could be considered that the two disparate images fell in the immediate neighborhood of the defect in the visual field, so that one of the two images came to lie on

14. Fischer, F. P.: Studien ueber Tiefenwahrnehmung, Arch. f. Physiol. **204:** 205, 1924.

the blind part of the retinas. We believe, however, that the horizontal disparity in our cases was much too little to warrant the possibility that one image could be perceived while the other one which lay just 10 seconds more toward the periphery was not perceived. But even if we believed that this were possible, we had to apply Hering's theory of the depth values of the retinal points. In figure 7 we had to assume that if the left rod was the nearer one, the two single images, as indicated in the drawing, would have a homonymous location on the right half of the cyclopean eye. Then the image which fell more to the right was more likely not to be perceived, in which case the decision of the observer was entirely dependent on the image which fell on the nasal

Fig. 7.—The hemianopic cyclopean eye if the left rod is the nearer one (on the left), and if the left rod is farther away (on the right). In the first case the image of the right eye is more likely to fall on the blind half of the retina, while in the latter case it is the image of the left eye which is probably not perceived.

half of the left eye. According to Hering, the nasal half of the retina has the depth value of relatively greater distance than the object fixed on. Therefore, the person who observed the rods in the way described would have decided that the left rod was farther away if he based his conclusion entirely on the described position of the images of the retina. In the opposite case, when the left rod was placed farther away and the same constructions were carried out, the observer would conclude that the rod was relatively nearer. It is seen that these considerations, which are vague, could be applied only if the hemianopic patient made incorrect statements exclusively, or said that the left rod was nearer when it was really farther away, and vice versa.

Another theoretical possibility that could be considered is the prevalence of the homolateral eye. As Tschermak [15] has shown, during the first few fractions of a second the influence of the homolateral eye is stronger than that of the contralateral eye. In a case of left-sided hemianopia, for instance, in which the left rod is fixed on and the right one is seen eccentrically, the influence of the right eye is stronger than that of the left eye. Therefore, especially during the first half of the second during which the rods are exposed, the right rod is projected farther away. This factor influences the localization in the same direction as does the curvature of the horopter. Therefore, it is likely that the prevalence of the homolateral eye accounts to a certain extent for our observations in hemianopia.

In our studies on cases of hemianopia we have seen that the fineness of binocular perception of relief may be very good in these cases. According to Heine, the ability of perceiving depth in the center of the visual field should be lost in cases of hemianopia without sparing of the macula. The parts of the visual field which were concerned in our experiments were slightly eccentric. Some tests were carried 1 degree, most of them 10 minutes, from the point of fixation. The question arose whether or not the acuity of depth perception could be tested for parts of the field which were any closer to the actual point of fixation than 10 arc minutes. Among the various methods that we used one gave very satisfactory results. Instead of placing the two rods side by side, we put one above the other, leaving a space of about 2 cm. between the ends of the rods. Normal persons tested with this method usually found it much harder to decide which rod was the nearer one, although one was of course nearer. They got the impression that they actually did not know which was the nearer one, and just guessed at it. The acuity of binocular depth perception, which is obtained thus by using the rods one above the other, is not quite as good as that obtained with the rods side by side. All of our hemianopic patients were tested with this method also. Each of them was found to be able to perceive the differences of depth in the vertical meridian which goes through the fovea almost as well as he did in the contiguous horizontal positions of his retinas. In all of these experiments the image of the target (the rod in our case) was much larger than the amount of disparity. That meant that both single images of the rod which was seen with horizontal disparity lay to some extent on the seeing parts of the retinas. The disparity of the part of the image which was perceived was apparently sufficient to lead to the perception of depth. Our observations were

15. Tschermak, A.: Studien ueber Tiefenwahrnehmung, Arch. f. Physiol. **204**: 232, 1924.

therefore not contradictory to the theory of Heine. They finally led us to a method which actually proved in our mind that Heine's conceptions were correct. This method consisted of making the rods as thin as possible—we chose fine black rods which subtended an angle of 1 arc minute—and placing them so far, one above the other, that the disparity amounted to about 2 arc minutes. If these experiments were carried out with the head of the patient kept fixed as exactly as possible and using only a half second as the time of exposure, some of our patients failed. They were not able to perceive depth if the disparity was larger than the size of the target, although they had perceived depth with the same targets if the disparity was smaller than the size of the targets. This phenomenon shows that the principle of Heine's theory is correct.

SUMMARY

The acuity of the binocular depth perception was tested in six cases of homonymous hemianopia without sparing of the macula. In all of the six cases the values obtained were within the limits allowed for normal persons. If the experiments were arranged in such a way that one of the two disparate images came to lie on the blind part, the other one on the seeing part of the retinas, then the patient failed. This is in accord with Heine's theory that in hemianopia without sparing of the macula the ability of perceiving the center of the field is lost. The eccentric perception of depth in cases of hemianopia shows the influence of the curvature of the horopter and of the prevalence of the homolateral eye.

# THE CENTRAL NERVOUS SYSTEM CONTROL OF THE OCULAR MOVEMENTS AND THE DISTURBANCES OF THIS MECHANISM *

HENRY ALSOP RILEY, M.D.
Professor of Neurology and Neuroanatomy, Columbia University
NEW YORK

Although this paper is principally concerned with the machinery provided for the ocular movements by the central nervous system and the disturbances in this mechanism, it would seem advisable to preface this discussion by a brief consideration of the action of the extra-ocular muscles, their innervation and the disturbances in this mechanism.

## CONTROL OF THE OCULAR MOVEMENTS BY THE EXTRA-OCULAR MUSCLES

The ocular movements are brought about by six muscles contained within the orbit that act either singly or in combination with each other in the production of deviations of the axis of the eyeball in all directions. These deviations take place as a result of volition or in response to reflex influences arising in other parts of the nervous system, conducted to the centers governing the eye movements by many different pathways.

The muscles concerned in the movements of each eye are the superior, external, inferior and internal rectus and superior and inferior oblique muscles. Each one of these muscles has a primary influence on the deviation of the axis of the eyeball. According to Wilbrand and Saenger,[1] from whose comprehensive presentation of the neurology of the eye much material has been drawn in the preparation of this paper, the action of each eye muscle results in the movement of the eyeball in one or another primary direction which may or may not be associated also with a subsidiary deviation.

The superior rectus muscle draws the eyeball upward and inward with a slight inward turning of the vertical meridian toward the nose. The internal rectus muscle is active principally in turning the eyeball inward toward the nose while the external rectus produces movement in the opposite direction. The inferior rectus muscle draws the eyeball downward and inward, turning the upper pole of the vertical axis away from the nose.

---

* Submitted for publication, June 30, 1930.

* Read before the Section of Ophthalmology of the New York Academy of Medicine, May 19, 1930.

1. Wilbrand, H., and Saenger, E. A.: Die Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 3.

The oblique muscles draw the eyeball in two directions and at the same time twist the vertical meridian of the eyeball either toward or away from the nose. The inferior oblique muscle draws the eyeball upward and inward and turns the upper pole of the vertical meridian away from the nose, while the superior oblique muscle draws the eyeball downward and outward and turns the upper pole of the vertical meridian

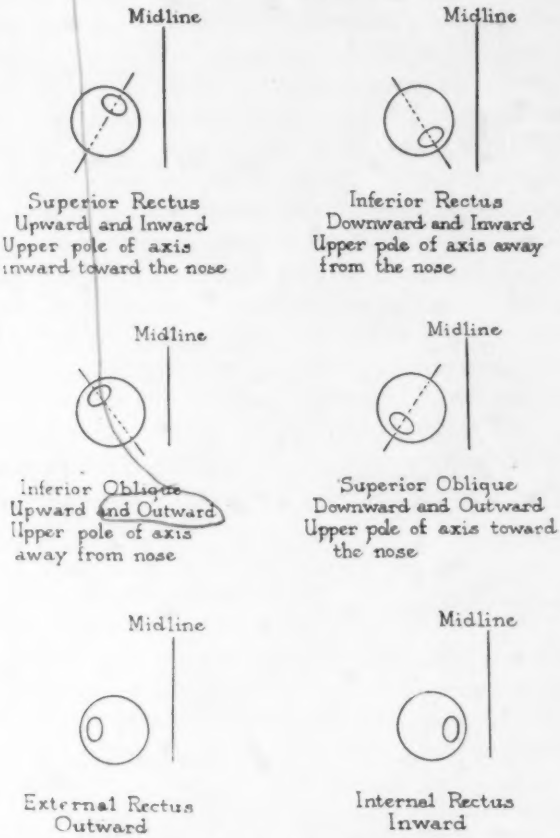



Fig. 1.—Primary deviations of the eye muscles. The right eyeball as viewed by the examiner.

toward the nose. In the combined action of the superior rectus and the inferior oblique muscles, the contradictory tendencies neutralize each other, and a direct upward movement results. Similarly, in downward movement the inferior rectus muscle draws the eyeball downward and inward, twisting the vertical meridian outward, while the compensating action of the superior oblique muscle in directing the eyeball downward and outward and twisting the vertical meridian toward the nose results in a direct downward movement of the eyeball.

In the majority of the movements of the eye, two or three muscles act together in producing the desired deviation: in adduction, the combined activity of the internal rectus, the superior rectus and the inferior rectus muscles; in abduction, the external rectus muscle acts alone; in upward movement, the superior rectus and the inferior oblique muscles; in downward deviation, the inferior rectus and the superior oblique

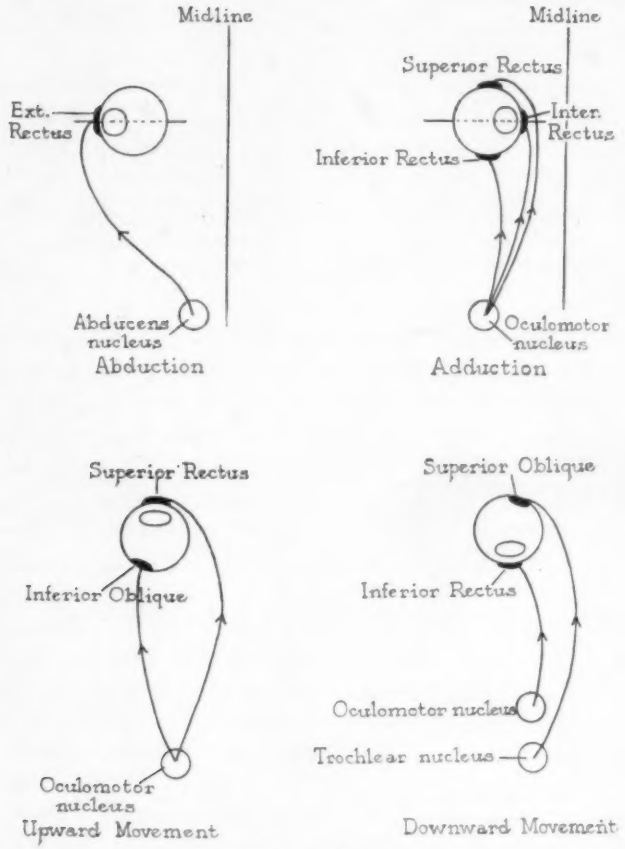



Fig. 2.—Movements of the right eyeball as viewed by the examiner.

muscles. Since the eyeballs are freely movable in any direction, and since muscles are supplied that are capable of drawing the eyeball in their primary directions, upward, downward, inward and outward, combinations of these movements may be brought about by the appropriate association of muscles producing the primary deviations so that the eyeball may be moved in any degree of obliquity, upward or downward, inward or outward.

From studies on the complicated interactions of other groups of muscles in the body made by Tilney and Pike [2] in their experimental study of the synergic action of the cerebellum, it would seem probable that in every movement, not only the dominant muscles are active but also the anatomically situated antagonistic muscles, the check elements that by finely graded cocontraction produce the exquisitely accurate and exact coordination that characterizes animal motion. If this is true with the ocular muscles, as it seems to be with the somatic musculature, practically all of the muscles of the eyeball should be in a state of greater or lesser tonal activity in all of the movements of the eyeball in order to provide the delicately balanced check component for the more active dominant element. This view is not in harmony with the work of Sherrington,[3] who found that if he cut the third and fourth cerebral nerves of one eye and then followed this by homolateral cortical stimulation, contraction of the contralateral external rectus muscle could be produced, and that the homolateral eye, the third and fourth cerebral nerves of which had been cut, then moved toward the contralateral side to the midline, but no farther. On this evidence and other facts, he based his conception of reciprocal innervation and the active relaxation of anatomically antagonistic muscles. Although we are not in any position to state categorical views in regard to synergic control, it would not seem impossible that movement of the eyes in any one direction would involve some tonic change in practically all of the muscles acting either as dominant or as check elements.

### DISTURBANCES OF THE CONTROL OF THE OCULAR MOVEMENTS PRODUCED BY DISEASE OF THE EXTRA-OCULAR MUSCLES

Interference with the ocular movements resulting from disease of the muscles themselves makes itself evident by weakness or paralysis of the particular muscle or muscles affected. This is manifested by a failure of the eye to move in the primary deviation produced by the muscle affected and also influences those other movements, in which the action of this affected muscle plays a secondary rôle. These disturbances are detected by careful examination of the volitional and reflex movements of the eyes by the various appliances in common use, particularly those connected with the detection of false and true images, since involvement of a single muscle is usually productive of a faulty fusion of the binocular fields of vision resulting from an unequal axial deviation of the eyes, the diplopia appearing in the movements of gaze in which the affected muscle would be chiefly operative. The principal processes responsible

2. Tilney, F., and Pike, F. H.: Muscular Coordination Experimentally Studied in Its Relation to the Cerebellum, Arch. Neurol. & Psychiat. **13**:289 (March) 1925.

3. Sherrington, C. S.: Experimental Notes on Two Movements of the Eye, J. Physiol. **17**:1994, 1927.

for such involvement of the eye muscles are rheumatic infiltrations, myositic deposits, trichinosis, hyaline and fatty degeneration of the muscle fibers of the extra-ocular muscles and other strictly muscular disturbances.

The possibility of mesodermic aplasia failing to produce the proper differentiation of one or another of the extra-ocular muscles from the somite must always be borne in mind, although the difficulty of differentiating this from a nuclear aplasia must be considered. Wilde [4] mentioned the possibility of congenital absence of abduction being due to the failure of the external rectus muscles to differentiate.

Hereditary ocular degeneration, ophthalmic abiotrophy, is discussed by Collins [5] who advanced the possibility that certain palsies of the eye muscles may be due to an inherent lack of viablity in the muscle tissue itself. He compared this condition with the muscle abiotrophies so well known in the myogenic dystrophies and atrophies. He quoted the well known words of Gowers in connection with the description of the muscle abiotrophies recognized by him as a "want of vital force in the structures involved so that they are unable to maintain their nutrition beyond full development and therefore gradually fail and degenerate . . . from imperfect life from abiosis in what may be designated abiotic atrophy or abiotrophy." He stated that degenerative changes have been found in the ocular muscles. No autopsies have been reported in cases of this kind and therefore full confirmation of this syndrome is lacking. He, however, mentioned one of these conditions as "hereditary ptosis," which may throw them into the group of the progressive external ophthalmoplegias, cases of which have not been extensively examined post mortem. Recently, however, one of these cases has been studied pathologically by Langdon and Cadwalader,[6] who showed mild but definite neuraxial changes. This would therefore throw some doubt on the possibility of a true myogenic abiotrophy. Further reference to these observations will be made in connection with the neurogenic ophthalmoplegias.

### CONTROL OF THE OCULAR MOVEMENTS BY THE PERIPHERAL OCULOMOTOR NERVES

The afferent stimuli that inform the various centers for coordination, associated movement and for the purpose of maintaining stereoscopic vision through deep muscle sensibility originate in the muscle and tendon

4. Wilde, A.: Congenital Absence of Abduction, Am. J. Ophth. **11**:781, 1928.

5. Collins, E. Treacher: Hereditary Ocular Degenerations Ophthalmic Abiotrophies, Tr. Internat. Cong. Ophth., Washington, D. C., 1922.

6. Langdon, H. M., and Cadwalader, W. B.: Chronic Progressive External Ophthalmoplegia, Brain **51**:321, 1928.

organs in the oculomotor muscles. These organs of deep muscle sensibility are affected by variations of tension in the oculomotor muscles, and stimulation of them gives rise to impulses that enter the brain stem and pass either to coordinating centers or to other end-stations within the central nervous system where their afferent impulses are made use of in coordinating the activities of the oculomotor muscles. Some of these impulses eventually reach the level of consciousness and contribute the feeling of tension and of strain that follows long continued use of the eye. The exact intra-axial course of these afferent sensory impulses from the oculomotor muscles is not well understood, but it is presumed that they join the tracts transmitting deep sensibility, contribute this information to the centers controlling ocular movements, and eventually establish connections with the visual cortex for the purpose of maintaining binocular and stereoscopic vision.

The efferent components form the three oculomotor nerves. The most cephalic of these oculomotor nerves is the third cerebral or the oculomotor nerve. The next more caudal is the fourth cerebral or the trochlear nerve and the most caudal is the abducens or the sixth cerebral nerve. The oculomotor nerve emerges from the oculomotor sulcus which is found on the mesial surface of the cerebral peduncle. At their exit from the midbrain, the radicles are collected together to form on each side a nerve of considerable size, which leaves the interpeduncular space, receives an investment of pia mater, passes forward in the subarachnoid space piercing this membrane and receiving an investment from it, and then enters the dura mater at the caudal end of the lateral wall of the pituitary fossa where the dural reduplication includes within it the cavernous sinus. The oculomotor nerve receives an investment from the dura mater and then divides into an upper and a lower group of fibers. The upper group of branches separate and supply the levator palpebrae superioris and the superior rectus muscles. The inferior group of branches traverse the lower half of the orbit to divide and supply branches to the inferior rectus and the inferior oblique muscles. The branches destined for the supply of the internal musculature of the eye pass forward along the outer aspect of the optic nerve and enter the ciliary ganglion from which branches are derived that enter the eyeball and supply the sphincter iridis and the ciliary muscle.

The fourth cerebral or trochlear nerves decussate in the roof of the isthmus caudal to the inferior colliculi, emerge from the superior medullary velum and pass on either side forward and around the lateral surface of the isthmus to enter the same fold of dura mater that is already occupied more dorsally by the oculomotor nerve. It proceeds forward in the lateral wall of the cavernous sinus, leaving the dura mater, from which it receives an investment, enters the orbit and passes along the upper wall of the orbit to enter the superior oblique muscle.

The sixth cerebral or abducens nerve emerges from the bulbopontile sulcus, a short distance from the midline, pierces the pia mater, enters the subarachnoid space, proceeds cephalad on the ventral surface of the middle cerebellar peduncle, and finally reaches the same destination that has been attained by the third and fourth cerebral nerves, the dura mater forming the lateral wall of the pituitary fossa. The sixth nerve then enters the substance of this lateral partition, being situated not in the lateral wall but between the two layers forming the cavernous sinus. It emerges from the interior of the sinus at the sphenoidal fissure, acquires a dural covering, enters the orbit and passes outward and forward to supply the external rectus muscle.

### DISTURBANCES OF OCULAR MOVEMENT PRODUCED BY INTERFERENCE WITH THE PERIPHERAL OCULOMOTOR NERVES

The peripheral nerves supplying the ocular muscles may be involved by any of the endogenous or exogenous toxic, metabolic or infectious disorders that are capable of exciting neuritic processes anywhere in the body. They also may be affected locally by any sort of pathologic process to which they may be exposed in their course in the subarachnoid space, in the wall of the cavernous sinus, in contact with the accessory sinuses of the nose, in the sphenoidal fissure or in the area of their distribution within the orbit itself.

The various neuritis-producing factors may affect any one of the oculomotor nerves, the so-called rheumatic disturbances, uric acid dyscrasia, the various forms of endogenous and exogenous intoxication, the infectious diseases, etc. The oculomotor nerves may be involved in the acute and chronic inflammations of the meninges. They are frequently involved by diseases that affect the lining of the sphenoidal fissure, where they may be encroached on by thickening of the periosteal covering of the bone. They are subject to injury by diseases of the blood vessels, aneurysms of the internal carotid artery or arteriovenous aneurysms involving the internal carotid artery and the cavernous sinus. They are also exposed to pressure from neoplasms arising from the base of the skull, from the meninges, the hyophysis and within the orbit. They are particularly prone to injury from trauma producing fractures of the middle cranial fossa. They are also subject to direct injury by penetrating wounds of the orbit.

The results produced by involvement of the oculomotor nerves in their peripheral course depend directly on the particular branches that are affected by the pathologic process. Any one or any combination of the three oculomotor nerves may be concerned to any extent. The distribution may be selective in that only a part of one nerve may be affected. In the case of the third cerebral nerve, the involvement may

be of a differential character, producing a weakness or paralysis of one or another of the muscles supplied by the nerve. The involvement of the oculomotor nerves in their peripheral course is made evident by weakness or paralysis of the muscle or muscles supplied by the particular nerve fibers that are affected. As in the case of disease of the oculomotor muscles themselves, the result is a loss of motility producing diplopia, the direction of which may be ascertained by the various means for the identification of paretic muscles. The diplopia appears when the person gazes in the direction in which the paretic muscle moves the eyeball. The determination of the extent of the involvement depends on the careful examination of the movements of the eyes in all directions.

A paralysis of the oculomotor nerve results in ptosis, an inability of the patient to move the affected eyeball upward, downward and inward, the eyeball is turned outward through the unopposed action of the external rectus muscle and the pupillary and ciliary reflexes are lost, the pupil being dilated. The chin is turned away from the paralyzed side in an effort to compensate for the abnormal deviation of the eye, and the frontalis muscle presents an excessive amount of activity through its attempt to raise the paretic or paralyzed upper eyelid.

Paralysis of the trochlear nerve results in a deviation of the head toward the shoulder and a sinking of the chin in order to obviate the effect of the double vision. When the gaze is directed downward, the paralyzed eye seems to lag somewhat behind the normal one with the production of a double vision. The image seen with the paralyzed eye is situated somewhat lower and with the upper end turned inward. Isolated palsies of the trochlear nerve are rather rarely seen but they have been described in fractures of the skull, endogenous and exogenous toxemias and also with the more chronic types of inflammation, particularly syphilis.

An isolated paralysis of the abducens nerve results in a failure of movement outward with the appearance of double vision. In order to obviate this diplopia, the head is often turned toward the side of the paralyzed muscle. A sensation of vertigo usually accompanies the double vision on account of the incorrect orientation of the person in space and in relationship to the object seen. The size of the deviation varies with the person and also with the duration of the paralysis and the direction of the gaze and also in part as the result of individual differences in muscle tone, the action of the antagonists and other anatomic conditions.

### NUCLEAR CONTROL OF THE OCULOMOTOR MUSCLES

The immediate motor control of the oculomotor muscles is exercised by the nuclei that give origin to the oculomotor nerves. The oculomotor nucleus is situated in the most caudal portion of the interbrain and the

cephalic portion of the midbrain, the trochlear nucleus is situated in the isthmus, and the abducens nucleus has its location in the metencephalon or pons varolii.

The oculomotor nerve rises from a nuclear mass that is situated in the caudal portion of the interbrain in the upper portion of the posterior wall of the third ventricle below the communication of the ventricle with the aqueduct of Sylvius, and is continued caudally in the central gray matter surrounding this canal. It extends downward to about the level of the middle of the inferior colliculus. The oculomotor nucleus consists of the following portions: a mesial collection of cells in the caudal wall of the third ventricle near the midline which is made up of small closely packed cells with few processes that stain less intensely than the adjacent cells and are similar in their characteristics to those giving rise to efferent vegetative fibers elsewhere in the central nervous system. This nucleus is called the nucleus of Edinger-Westphal and is considered to be a portion of the vegetative nervous system. Brouwer[7] reviewed the various ideas that have been expressed concerning the activity of the Edinger-Westphal nucleus, quoting the observations of Tsushida who does not believe that the Edinger-Westphal nucleus belongs to the essential oculomotor apparatus. He stated that the Edinger-Westphal nucleus is considered by some authors to have nothing to do with the pupillary reaction, and that Cajal was not able to trace fibers from the Edinger-Westphal nucleus into the oculomotor nerve. He considered the phylogenetic history of the nucleus and reported that this nucleus is found in mammals, birds and reptiles, but that it has no representation in fish. As a result of his own anatomic and clinical investigations, Brouwer believed that the Edinger-Westphal nucleus is the autonomic nucleus that gives rise to the fibers that supply the internal muscles of the eye, namely, the sphincter iridis and the ciliary muscles. Another mesially placed group of larger nerve cells dorsal to the Edinger-Westphal nucleus constitutes the nucleus of Darkschewitsch, which, although in intimate relationship with the nuclei of the oculomotor complex, is not supposed to have anything to do with oculomotor function.

The chief nucleus is a bilateral columnar series of cell groups extending from the caudal limit of the third ventricle to about the middle of the inferior colliculus. The nucleus is divided into a number of constituent segments, the identity of which varies according to the views of different investigators. Bernheimer,[8] who investigated the nucleus

7. Brouwer, B.: Klinisch anatomische Untersuchung über den Oculomotoriuskern, Ztschr. f. d. ges. Neurol. u. Psychiat. **40**:152, 1918.

8. Bernheimer, S.: Experimentelle Studien zur Kenntnis der Innervation der inneren und ausseren von Oculomotorius versorgten Muskeln des Auges, Arch. f. Ophth., 1897, p. 44.

after serial extirpations of the muscles of the eyeball, came to the conclusion that the cephalocaudal sequence of nuclear control was as follows: the levator palpebrae superioris, the rectus superior, the internal rectus, the inferior oblique and the inferior rectus muscles. Brouwer established the same sequence for the nuclear segments. The portion of the nucleus that supplies the internal rectus is placed by Brouwer at about the middle of the chief nucleus. He considered it to be divided into two parts: a lateral subdivision and a more median portion. This median segment fuses across the midline with its fellow of the opposite side to form the nucleus of Perlia which is responsible for the movement of convergence, a true center for oculomotor movement. He stated that the nucleus of Perlia is the last portion of the oculomotor nucleus to differentiate, and that its appearance in the primate series accords with the inception of the function of convergence. The lateral portion of the internal rectus segment, he believed, controls the action of the internal rectus muscle in lateral deviations.

LeGros Clark [9] expressed the belief that the segmentation of the oculomotor nucleus is arranged dorsoventrally and not cephalocaudal, and that even in the nucleus the grouping of the cellular elements is according to function and not in accordance with the muscles supplied. He believed that the inward movement of convergence is controlled by the paramedian group, the nucleus of Perlia; that upward movement is governed by the dorsal segment and downward movement by the ventral portion of the nucleus. This differentiation of the nucleus is not in accord with the results of other investigators.

Other segregations of function have been suggested, but the latest and best supported evidence seems to favor the division of the nucleus according to the idea of Bernheimer and Brouwer. The nucleus is embedded ventrally in the fibers of the posterior longitudinal fasciculus. The nucleus of each side gives rise to many fibers that pass across the raphe to the nucleus of the other side. These fibers link together the various parts of the nucleus that supply the muscles of the eyeball in order to correlate their action in the intricate movements of conjugate deviation. The actual number of fibers that take part in this decussation can be seen to increase progressively as the organization of the brain becomes increasingly complex in the ascending phyletic scale and seems to be in direct relationship to the extent and complexity of the development of conjugate movement. This has been graphically pointed out in a recent publication by Tilney.[10]

---

9. Clark, LeGros, quoted by McCowan and Cook, L. C.: Oculogyric Crises in Chronic Epidemic Encephalitis, Brain **51**:285, 1928.

10. Tilney, F.: The Brain from Ape to Man, New York, Paul B. Hoeber, Inc., 1928.

The fibers that are destined to become root fibers of the oculomotor nerve emerge from all parts of the nucleus and proceed ventrally into the tegmentum, being aggregated into larger and larger nerve fascicles, passing either through or around the nucleus ruber and through the mesial portion of the substantia nigra to emerge as discrete radicles from the oculomotor sulcus situated on the mesial surface of each crus cerebri.

The trochlear nucleus is situated in the dorsomesial angle of the tegmentum of the isthmus encephali embedded about equally in the gray matter surrounding the aqueduct of Sylvius and the mass of fibers form-

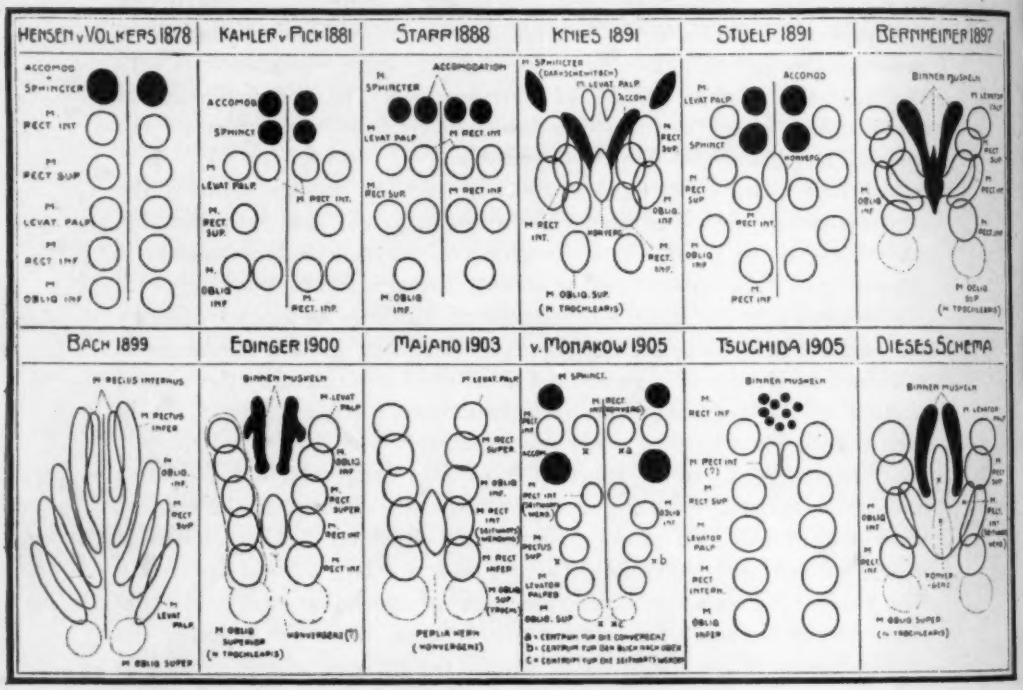



Fig. 3.—Morphologic segmentation and functional localization of the oculomotor nucleus according to various investigators. (From Brouwer, B.: Klinisch anatomische Untersuchung über den Oculomotoriuskern, Ztschr. f. d. ges. Neurol. u. Psychiat. **40**:152, 1928.)

ing the posterior longitudinal fasciculus to which it furnishes, and from which it receives, many fibers. There are no commissural fibers joining the nucleus of one with the nucleus of the other side. Emerging from the various aspects of the nucleus, the rootlets of the nerve become collected into a nerve of moderate size, which proceeds backward in the boundary between the central gray matter and dorsal prolongation of the tegmentum until it reaches the roof plate, the superior medullary velum, where it turns mesially, decussates with its fellow of the opposite side

and emerges from the dorsal surface of the neuraxis. Wilbrand and Saenger claimed that the decussation of the trochlear nerve is incomplete, stating that the caudal part of the nerve completely crosses but that the cephalic portion, that part which is in closest relation with the caudal portion of the oculomotor nucleus supplying the rectus inferior muscle, does not pass across the midline.

The abducens nucleus is situated in the middle of the metencephalon. It lies partly within the dorsal portion of the tegmentum and partly within the subventricular gray matter, lateral to the posterior longitudinal fasciculus with which it is connected by many fibers. The facial nerve in its ascending portion lies on the medial aspect of the abducens nucleus, surrounds the cephalic pole of the nucleus and then descends on its lateral aspect leaving it to enter on its emergent course. The fibers of the abducens nerve emerge from all parts of the nucleus, but chiefly from its lateral aspect, and are collected together to form the nerve that passes ventrally and somewhat caudally through the tegmentum and basis to emerge from the bulbopontile sulcus lateral to the medullary pyramid.

## BRAIN STEM CENTERS CONTROLLING THE OCULAR MOVEMENTS

From this point on, the description of the mechanisms that control ocular movement enters a realm in which knowledge is greatly interspersed with suppositions, for much of the data accumulated in regard to the movements of the eyeballs is conjectural and not supported by authenticated histologic evidence. A great deal of the information has been obtained by the examination of clinical material afflicted with a variety of oculomotor disturbances. In many instances, autopsy material has been presented in evidence supplying information of a negative character, and but little of it possesses positive localizing value. From the data that have thus accumulated, certain formulations have been made as to the existence of centers that distribute fibers to certain nuclei or parts of nuclei that are concerned with conjugate movement in one or another direction, these centers being in turn under the control of impulses descending from suprasegmental sources.

## THE CENTER FOR LATERAL DEVIATION OF THE EYES

The muscles of the eyes act together in such a way as to maintain parallelism between the visual axes of the eyes, thus obviating double vision which is occasioned by the incidence on nonhomonymous retinal points, of light rays reflected from the object seen. The position of the eyeball is dependent first of all on the relationship between the eyeball and the adnexa, namely, the optic nerve, the blood vessels of the eye, the form and size of the eyeball and the connective and bony tissues of the orbit,

and secondly on the balanced action of the muscles attached to the eyeball. The mechanical factors are individual in each case and are counterbalanced by a greater or lesser activity in the various eye muscles as conjugate or fused vision develops.

For many years the abducens nuclei have been considered the pacemakers of lateral movements of the eye, the nucleus itself being looked

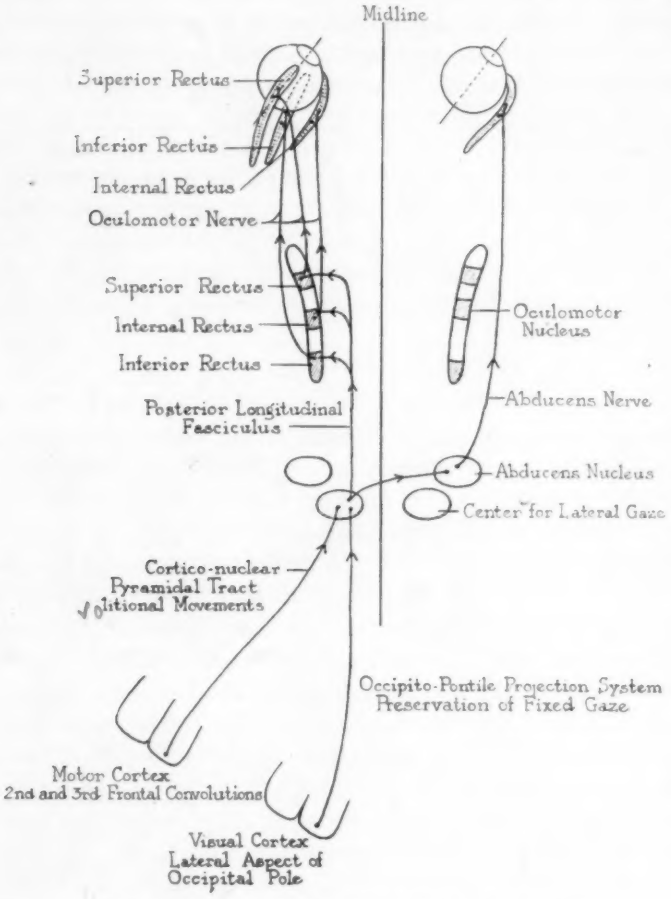



Fig. 4.—Mechanism for lateral deviation of gaze.

on as the center for these deviations. In this movement the external rectus muscle innervated by the abducens nucleus of one eye is associated with the internal rectus, superior rectus and inferior rectus muscles of the opposite eye, all of which are supplied by the oculomotor nucleus. It is obvious that some connection must be established between the action of these two nuclei. This may be accomplished either by the pyramidal tract fiber splitting and passing to both nuclei or by the

intermediation of a center that shall send fibers to both the abducens nucleus of one side and the appropriate segments of the oculomotor nucleus of the other side. The latter is the generally accepted explanation, and the center for lateral gaze is predicated to exist close to the abducens nucleus, sending fibers directly to the homolateral abducens nucleus and to the contralateral oculomotor nucleus by the posterior longitudinal fasciculus. Certain instances of loss of movement of the homolateral eye outward with retention of the conjugate movement of the contralateral eye inward have been described in which the abducens nucleus is completely destroyed, thus showing that the center for this lateral movement cannot be situated directly in the nucleus itself, although other pathologic evidence would indicate that this center must be close to the nucleus. In other instances there may be a paralysis of lateral deviation without any demonstrable lesion of the sixth nerve nucleus, but this evidence is not conclusive, since suprasegmental lesions may produce loss of lateral deviation without the nuclei or centers of lateral movement being involved. The conclusion to be reached is that the center is not identical with the abducens nucleus but is situated in the near vicinity.

The disturbances in lateral conjugate movement as a sequel of involvement of the centers governing this movement may result from almost any pathologic process involving the appropriate portion of the brain stem. In the absence of other focal evidence, it is difficult and at times impossible to determine whether involvement of the supranuclear pathways or the brain stem centers is responsible for paralysis of gaze. The presence of other symptoms indicating a focal involvement of the brain stem, such as a facial palsy affecting the side of the face toward which the gaze is paralyzed, would make a definite localization possible. Bilateral loss of conjugate gaze may be found. This is usually due to midline pontile lesions, for only in this vicinity is it possible to involve the centers of both sides by a single lesion of moderate size. In this instance, the activity of the internal recti muscles in the movement of convergence is maintained.

### THE CENTER FOR VERTICAL DEVIATION OF THE EYES

The evidence for the existence of this center is not so well authenticated as that for the center for lateral deviation. It has long been known that lesions of the collicular plate or pathologic processes arising in its vicinity may give rise to paralysis of vertical gaze, the so-called syndrome of Parinaud.[11] This syndrome may be further subdivided into paralysis of the elevators or of the depressors or both combined. These

11. Laignel-Lavastine, and Bernal, Pierre: Encéphalite aigue avec syndrome de Parinaud précoce compliqué et transitoire, Rev. neurol. **1**:479, 1929.

movements are produced by the interaction of the various muscles supplied by the oculomotor and trochlear nuclei and have already been described. In addition the movements of the two eyes together must be coordinated by a simultaneous stimulation of appropriate muscles

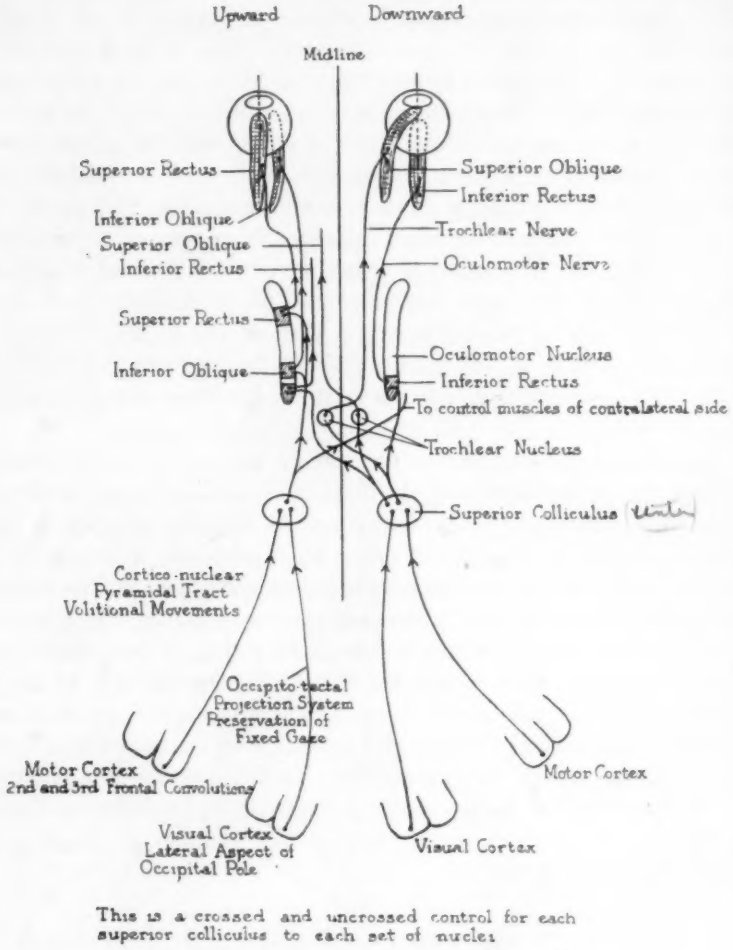



Fig. 5.—Mechanism for vertical deviation of gaze.

supplied by the oculomotor and trochlear nuclei together with the accompanying movements of the upper eyelid and frontalis muscle.

The loss of conjugate vertical gaze has been associated by many authors with lesions in the vicinity of the collicular plate, and much pathologic evidence has been adduced to support this view. Neoplasms of the epiphysis almost regularly show this disturbance in eye movement. It is also found with considerable regularity in the thalamic syndrome

of Déjerine-Roussy when the pathologic process invades the subthalamus sufficiently to involve the subthalamic contingent of the aberrant pyramidal tract that controls vertical movement. It has been described also in a rather miscellaneous group of clinical conditions, all of which, however, show evidence of involvement of the brain stem in the region of the diencephalomesencephalic junction. It has also been described as a distant pressure sign in tumors of the brain, in disease located in the posterior cranial fossa, in conditions involving the petrous portion of the temporal bone and in other indiscriminate combinations.

In placing the responsibility for paralysis of vertical gaze of either elevation or depression or both in the collicular plate, particularly in the superior colliculus, it is difficult to eliminate the factor that may be represented by direct pressure by the lesion on the oculomotor nucleus that lies directly ventral to this region in the central gray matter of the midbrain. In certain instances, however, paralysis of vertical gaze unassociated with pupillary changes and with no loss of convergence has been seen, and it would be difficult to conceive of pressure on the oculomotor nucleus being so selective in action as to affect only the muscles concerned with vertical gaze and spare the nuclei of Edinger-Westphal and of Perlia. The fact that in the vertical movements of the eyes the trochlear nucleus is associated with the muscles supplied by the oculomotor nucleus would also bespeak the necessity for a coordinating center for this movement, apart from the oculomotor nucleus. It would therefore seem probable that the superior colliculus may be considered to be either a relay station in the suprasegmental pathway for the control of vertical gaze or in reality a subcortical center under the influence of suprasegmental structures that serve as a distributing station for the voluntary or reflex patterns involved in vertical gaze. From clinical observation, it appears that with long-standing lesions of the collicular plate a later return of a considerable degree of vertical gaze takes place, thereby indicating some substitution in this mechanism. Permanent paralysis of vertical gaze, on the other hand, would seem to indicate the probability of a lesion in the cephalic extremity of the oculomotor nucleus producing destruction of the cells governing the upward and downward movement of the eyes.

Hiller (quoted by Wilbrand and Saenger) collected forty-seven cases of paralysis of vertical gaze, in twenty-six of which the patients were found to be suffering from a paralysis of the movements of the eyes upward. In five cases only was the downward gaze paralyzed, and in sixteen cases both the upward and downward movements of the eyes were involved. In fifteen cases, some sort of a disturbance in lateral movement was associated with paralysis of vertical gaze. Bilateral paralysis of vertical movement has also been described as a result of lesions that affect only one of the superior colliculi, and it is rather

the rule to find in lesions of the superior colliculus a symmetrical paralysis of the upward and downward movement in both eyes. The rich decussation of fibers between the two superior colliculi amply accounts for this. Sufficient clinical evidence supported by autopsy observations has accumulated to warrant the assumption that the superior colliculi control the vertical movements of the eyeballs either through the pres-

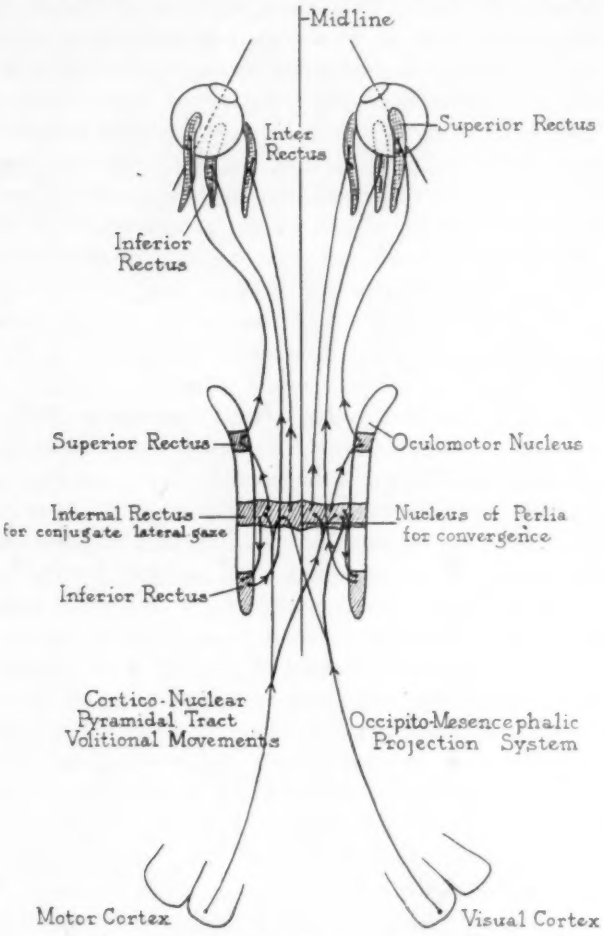



Fig. 6.—Mechanism for convergence.

ence of a center or through fiber pathways that pass through the colliculi. The former possibility would seem to be more probable since so far as could be determined by microscopic examination no pathologic process could be demonstrated in the oculomotor nucleus itself. There is some further physiologic evidence adduced by stimulation of the superior colliculus in which movements were found in various directions but more predominantly in the vertical plane.

### THE CENTER FOR THE CONVERGENCE MOVEMENT OF THE EYEBALLS

As a result of Brouwer's study of the oculomotor nucleus, he predicated a center for convergence in the midline nucleus of Perlia, a single nucleus made up of the mesial portion of the segment of each oculomotor nucleus that presides over the activity of the internal rectus muscle. There is considerable clinical evidence to support the view that there is a special nucleus for the convergence movement of the two eyeballs. Many patients have been reported to show complete failure of convergence. This is readily demonstrable in many patients suffering from epidemic encephalitis, and may be considered as one of the characteristic lesions of this disease. Wilbrand and Saenger stated that true convergence paralysis is rare, but their material was collected before the large group of cases of persons suffering from epidemic encephalitis was gathered together, in whom this palsy has been found to be relatively common. In these patients, the lateral associated movements of the eyes are perfectly maintained, the internal recti of each eye acting in lateral conjugate gaze, but either partially or totally failing to produce the convergence movement of the eyeball. Clinical cases have also been reported that show a paralysis of lateral gaze with inability to move the eyes to one side or to the other or to both sides in which convergence is still retained. These clinical facts would make it probable that there is a collection of nuclear matter somewhere in the midbrain that is responsible only for the control of the internal recti muscles in convergence, separate from the internal rectus segments that are concerned with the movements of lateral gaze. Wilbrand and Saenger expressed the belief that this nucleus is located in some part of the central gray matter in the vicinity of the superior colliculus. Brouwer's work demonstrated that with the appearance in the phyletic scale of the ability to converge, the only new addition to the oculomotor complex is the medianly placed nucleus of Perlia, and on this evidence he based the assumption that this nucleus is in reality the center for convergence. So far as can be determined, no definite pathologic evidence has been brought forward to substantiate this view, but it has received almost universal acceptance.

### THE CENTER FOR THE MOVEMENT OF DIVERGENCE OF THE EYEBALLS

The evidence for the assumption of a center for divergence is not nearly so definite as that for the center for convergence. It is believed by many that there is no separate center for divergence, and that under ordinary circumstances divergence is merely the result of a relaxation of convergence of the eyeball. Apparently there have been no reports of

the voluntary ability of any person to diverge both eyes at the same time. Cases of unilateral abduction have been described, but reference to active voluntary divergence of the eyeball does not appear in the literature investigated.

Straub [12] expressed the belief that there is a center for divergence that establishes a system by its control over the two abducens nuclei and acts in a manner antagonistic to the convergence system that has been so definitely established.

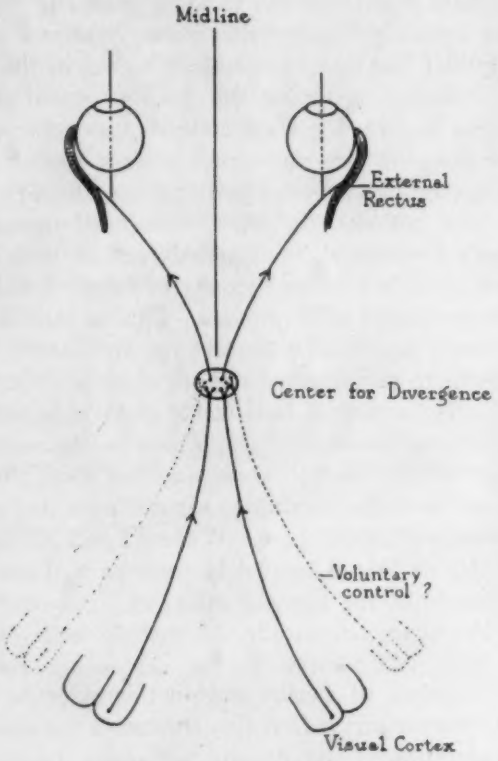



Fig. 7.—Mechanism for divergence.

Dunphy [13] described paralysis of divergence as an actual oculomotor defect, and stated that it is characterized by a homonymous diplopia that does not appear until the person looks at an object situated more than 20 feet from the eyes, beyond which point a divergence palsy makes its appearance. There is no diplopia at the ordinary reading distance, and all of the other ocular movements may be normal. I recently saw

12. Straub, quoted by Wilbrand and Saenger (footnote 1).
13. Dunphy, E. B.: Paralysis of Divergence, Am. J. Ophth. **11**:298, 1928.

a patient who complained of a weakness of divergence, describing the defect in vision by saying that the moon when viewed with the two eyes without glasses appeared double, and that all objects beyond about 20 feet were somewhat obscured. This was to a considerable extent corrected by the use of glasses. There were no abnormalities in the other movements of the eyes.

The pathway by which these various centers are connected and also by means of which the three oculomotor nuclei interchange fibers is supplied by a bundle of fibers that lies at the dorsomesial angle of the tegmentum of the midbrain, isthmus and rhombencephalon. This bundle is called the posterior longitudinal fasciculus. Its constituents number, not only the internuncial fibers passing between the various centers already mentioned and between the various nuclei of the oculomotor apparatus, but also the fibers that are concerned with the reflex activities of the eye movements, these being chiefly represented by the trigeminal, the acoustic and the vestibular contributions. It also serves to transmit the impulses that are received from suprasegmental sources that control the associated, synergic and voluntary control over the oculomotor movements.

As was pointed out in the preliminary statement concerning the activity of the muscles, the only movement of the eyeball that is produced by one muscle acting alone is that of abduction of the eyeball. In all other movements, whether upward, downward or inward, two and three muscles are associated. Adduction is controlled entirely within the domain of the oculomotor nucleus itself, but in all other movements the other nuclei collaborate with the oculomotor nucleus. The association of these muscles in the various movements of the eyes is brought about by fibers passing over the posterior longitudinal fasciculus between the three oculomotor nuclei.

### THE REFLEX MOVEMENTS OF THE EYEBALL

Important connections are established between the eighth cerebral nerve and the oculomotor mechanism. This is true for both divisions of the auditory nerve, the cochlear or acoustic division and the vestibular division. The connection between the acoustic division and the oculomotor mechanism is provided principally to produce the lateral movements of the eyeballs and is represented by the peduncle of the superior olivary nucleus that passes dorsally and mesially toward the vicinity of the abducens nucleus. In all probability this connection does not actually terminate in this nucleus itself, but in the closely adjacent center for the lateral movements of the eyes. A similar mechanism is provided through the posterior longitudinal fasciculus for the identification and localization of sources of sound situated above or below the plane of the

eye. These connections result in a rapid reflex turning of the eyes upward or downward.

In a connection with the vestibular division, impulses pass chiefly from the nucleus of Dieters, one of the reception nuclei of the vestibular division of the auditory nerve, directly across the dorsal portion of the tegmentum, and enter the posterior longitudinal fasciculus of both sides. These fibers end in the centers provided for conjugate gaze laterally and vertically, and serve to direct the eyes in one or another direction as the result of disturbances in equilibrium. The connections are manifestly bilateral and associated, since in disturbed conditions of the labyrinth or Deiters' nucleus or its fibers, the movement in almost all instances affects both eyes and produces rapid conjugated oscillations of the eyeballs to one side or another, upward or downward or in a rotary direction. In many of the lower forms fibers can be seen passing directly from the vestibular nerve into the posterior longitudinal fasciculus and thus to the centers concerned with ocular movement or to the individual nuclei. In the higher forms, however, this connection is probably not direct but is interrupted in the vestibular nuclei.

According to Bartels,[14] the secondary fibers from the vestibular complex pass over the posterior longitudinal fasciculus to the various oculomotor centers or nuclei and connect them. According to his description, it is uncertain from which vestibular nuclei these fibers are derived and to what extent they are crossed and uncrossed. Certain authors, as stated by Bartels, maintain than von Bechterew's nucleus contributes chiefly to these oculomotor movements. By others, however, it is claimed that Schwalbe's nucleus is mainly concerned in these connections. Bartels believed that Deiters' nucleus is chiefly responsible for these movements, and he stated that the rotary movements of the eyeballs are served chiefly by fibers from the caudal portion of Deiters' nucleus, that the vertical movements are controlled by fibers that arise in the cephalic portion of the nucleus, while the lateral movements are produced by the middle portion of the nucleus. He stated that the movements are so perfectly coordinated that the presence of a center providing for this perfect coordination must be accepted. Bartels believed that each canal probably has its own separate intra-axial center that distributes the stimuli caused by the movement of the sensitive hairs contained within the canal. The preservation of some of these reflex movements with a loss of others shows that they cannot, or in all probability do not, pass over the same pathway. In all likelihood also, all of the canals are connected with all of the muscles, for

14. Bartels, M.: Die Reflexbahnen für die Ohr-Augenbewegungen, Arch. f. Ophth. **117**:538, 1926.

after unilateral labyrinthine extirpation allowing sufficient time for the stormy sequels to subside, stimulation of the remaining labyrinth causes movement of both eyes in all directions but of unequal strength. This perhaps may be interpreted as evidence of the action of the control of both vestibular nerves over oculomotor centers rather than the distribution of fibers to all of the nuclei without the intermediation of a center.

Bartels stated that nystagmus persists after the removal of the hemispheres, cerebellum and section of the trigeminal nerve and all of the eye muscles except the external rectus. In the neurologic acceptance of the term "vestibular nystagmus," two phases are usually described. The slow phase is believed to be due to the irritative influence causing a deviation of the eyes in one or another direction and the rapid phase that brings the eyeballs back to the point of fixation, and is interpreted as being due to impulses from suprasegmental sources concerned in the preservation of gaze in one direction or another which is interfered with by the irritative activity of the lesion producing the slow deviation away from the point of fixation. This would not be consonant with the results of Bartels' investigation in which nystagmus, as he describes it, persists after an almost complete isolation of the oculomotor mechanism and its reduction to the movement of the external rectus muscle alone. It may be possible, however, that the mechanism is not the same in the lower animals that were used for laboratory experimentation as in the human being, and the rapid phase in man may still be considered as due to the influence of suprasegmental structures on the disturbed oculomotor mechanism.

The trigeminal reflex connections are those chiefly concerned with the movements of the eyeball in connection with sensory stimulation of the skin on the face. These movements are mediated through secondary fibers that leave the trigeminal nucleus and pass to the centers producing the coordinated movement of the eyes. Similar impulses are also transmitted to the oculomotor nuclei in connection with tactile and particularly painful stimuli connected with the body surface by which the location of the stimulating object and its character may be immediately determined by the reflex turning of the eyes to the source of the irritating sensation.

*(To be Continued)*

# A MODIFICATION OF GONIN'S SURGICAL METHOD OF TREATMENT OF DETACHMENT OF THE RETINA*

C. E. FINLAY, M.D.
HAVANA, CUBA

The brilliant results obtained in recent years by Gonin [1] and some of his followers with his new surgical treatment for this generally intractable disease have revived hopes of being able to deal with it more satisfactorily, by this or by some similar method of treatment.

At the beginning of 1929, being much interested in a case of traumatic detachment which occurred in a university athlete, and which proved refractory to the usual treatment first at the hands of a confrère and later of myself, I decided to make an effort to obtain a permanent cure along the lines recommended by Gonin.

A careful study of the method, as found in the recent literature,[2] excited in me certain, possibly only theoretical, objections. The most important of these was that the introduction of a cautery point through the different coats of the eye into the vitreous might establish, by a consequent prevention of primary union, a permanent fistulous communication between the vitreous, preretinal, subretinal, subchoroidal and external lymphatic spaces, which might not necessarily be beneficial. It appeared to me that a surer and safer result might be obtained by opening temporarily the subretinal space, letting out the displacing fluid and thus permitting the retina to reapply itself to the choroid. This should be followed by a superficial cauterization of the sclera in the immediate vicinity of the scleral wound, respecting carefully the wound edges themselves as their cauterization might interfere with primary union, with the object of provoking an adhesive chorioretinitis which would bind down the detached retina. In the case at hand, only a doubtful rupture could be made out at the site of the detachment, below and outward; therefore I decided to operate at this point. The technic of the operation was as follows:

I first made a meridional incision through the conjunctiva and layers of Tenon's capsule down to the surface of the sclera; I bared

* Submitted for publication, June 10, 1930.

1. Gonin, J.: Rev. gén. d'opht., August, 1923; Swiss Ophth. Soc., June, 1926; Ann. d'ocul. **164**:816, 1927; Arch. d'opht. **45**:554, 1928.

2. Sourdille: Ann. d'ocul. **161**:627, 1924; Arch. d'opht. **41**:694, 1924. Lindner: Ophth. Soc. Vienna, Dec. 18, 1928. Dubois, H.: Ann. d'ocul. **166**:81, 1929. Arruga: Arch. de oftal. hispano-am. **30**:581, 1929.

this carefully, and, keeping both edges of the incised tissues apart with small squint hooks, I then made a series of longitudinal cuts with a Graefe knife in the same groove in the sclera till it was perforated and retinal fluid exuded through the wound. I then proceeded to cauterize the scleral surface superficially, about 0.5 mm. from the edges of the wound, avoiding these carefully. I next sutured the conjunctival wound and applied a pressure bandage, which I did not remove for several days, during which time I kept the patient in bed. As will be seen in the clinical history of this case, the result was most satisfactory.

In other cases I made the conjunctival incision at right angles to the scleral, a detail which I do not consider of any importance. In one case in which a rupture could be seen above and internally and a large detachment below and externally, I made a double operation at both sites.

The following are the clinical histories of the patients I have treated according to this method.

## REPORT OF CASES

CASE 1.—R. G., aged 22, a university student, consulted me first about his eyes on April 11, 1929, for visual disturbances arising from an accident while he was playing football two months before. After the accident he was unconscious for fourteen days, without any motor or speech disturbances. During the first days, a lumbar puncture revealed blood, which later disappeared. There was no external hematoma. A roentgenogram proved negative for fracture. On recovering consciousness, the patient complained of poor sight, for which he consulted an ophthalmologist, who diagnosed hemorrhagic neuroretinitis (?). Under his treatment there was no improvement in the sight.

There was some bilateral blepharospasm (L > R). The skin of the nose showed burns from diathermy. Both pupils were contracted, but reacted normally to light. The right fundus was normal. The left fundus showed a retinal detachment below and outward. The visual field in the left eye was defective above.

I put the patient to bed and kept him there for two months, giving him daily injections of pilocarpine. At the end of two months there was no improvement, the sight in the left eye being 6/200, with no vision above and internally, the ophthalmoscopic lesions remaining unchanged.

On July 2, the operation described was performed.

There was immediate improvement in vision, and his visual field on July 27, 1929 (chart 1), showed a loss of the nasal field and part of the upper field. Central vision gradually improved till on May 27, 1930, it was 20/15, the visual field being practically the same as ten months before (chart 2).

CASE 2.—J. A., aged 18, entered Mercedes Hospital on July 16, 1929, with a history of having been hit in the left eye with a ball two and one-half years before. The right eye was normal, with a vision of 20/20. The left eye showed a large detachment involving almost the whole retina, the vision being 3/200.

An operation was performed on August 19, on the same lines as in the preceding case. The result was unsatisfactory.

Case 3.—V. L. P., aged 52, was referred to me on Oct. 17, 1929, by Dr. Anton of Camagüey, Cuba. He first saw the patient on August 28, and made the diagnosis of detachment of the retina in the left eye below with a whitish discoloration above. On September 5, he performed a posterior sclerotomy below and outward between the inferior and the external recti, the patient afterward being put to bed;

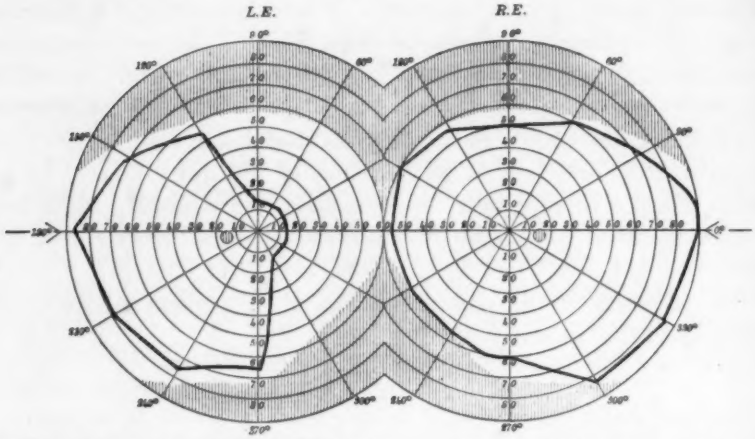



Chart 1 (case 1).—Visual fields taken on July 27, 1929, showing a loss of the nasal field and part of the upper field.

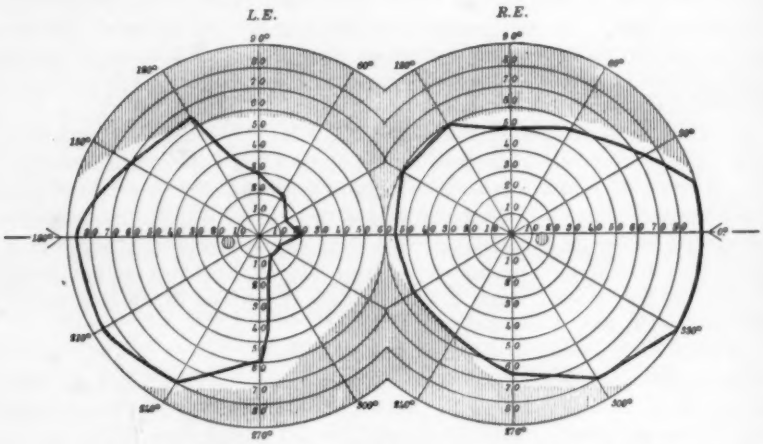



Chart 2 (case 1).—Visual fields taken on May 27, 1930.

this was followed by five daily subconjunctival injections of gelatinized acoinized serum. Two days later the detachment had disappeared. On September 20, a punctiform cauterization of the sclera below and outward was performed. On September 27, the patient was discharged cured, with a complete visual field. A week later a relapse occurred, with a reproduction of the detachment above and in and below and out.

On examination I found in the left eye a detachment of the retina below and out with a retinal rupture above and in. The vision of the right (normal) eye was 20/20; that of the left eye, 10/200. The visual field showed a defect above and internally (chart 3).

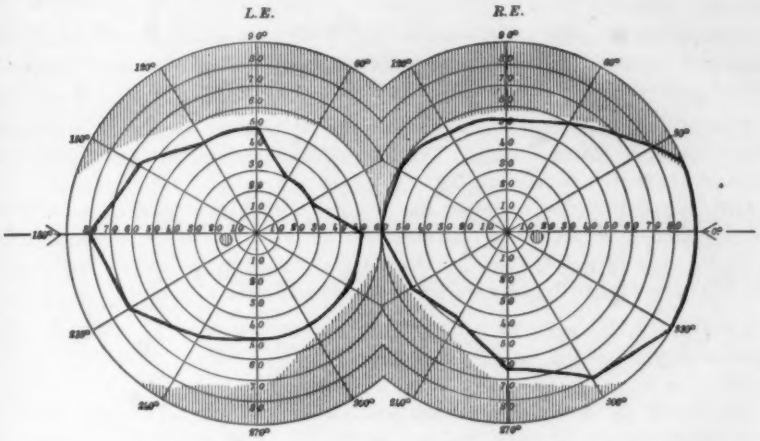



Chart 3 (case 3).—Visual fields taken on Oct. 17, 1929, showing a defect above and internally.

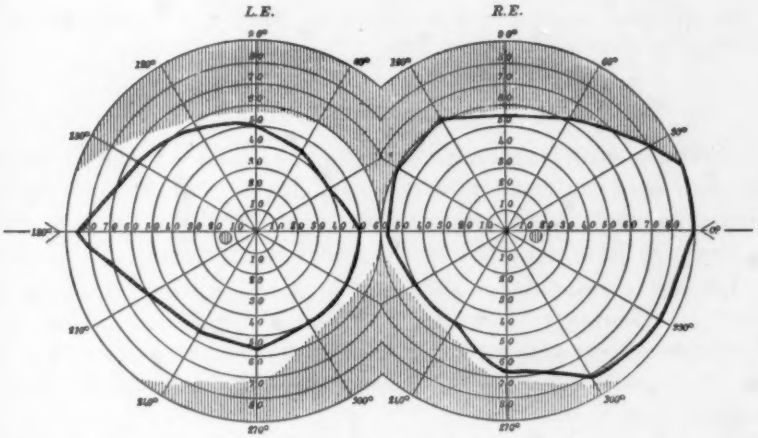



Chart 4 (case 3).—Visual fields taken on Nov. 12, 1929; the defect is less pronounced.

On October 26, I performed a double operation above and internally and below and externally. On November 12, vision had improved to 15/200, and the defect in the visual field was less pronounced (chart 4). This patient had suffered from glycemia, which had improved under treatment.

CASE 4.—A. R. V., aged 80, entered Mercedes Hospital on Feb. 2, 1929, with an old detachment of the right eye, which was aphacic and which had become blind more or less suddenly several months after a successful extraction by a confrère, and a hypermature cataract of the left eye. I performed a combined extraction, and on March 13 the patient was discharged with a vision of 20/200 and a thin secondary cataract.

The patient returned in November with a detachment of several days' duration and a vision of 2/200. On November 9, I operated on him according to the method outlined. The outcome was unsatisfactory.

CASE 5.—F. L. R., aged 17, first came to Mercedes Hospital in June, 1927, with congenitally luxated lenses. After discissions and extractions in both eyes, vision in the right eye was 20/200.

On Dec. 5, 1929, he reentered the hospital with a retinal detachment below and a vision of 20/100. On December 27, the usual operation was performed.

At the time of writing his eye showed a detached retina below, well limited, with a vision of 20/70 and a defect in the visual field above.

CASE 6.—M. R., aged 53, entered the hospital. On Oct. 11, 1929, an intracapsular extraction was performed on the right eye with a resulting vision of 15/200.

On March 28, 1930, he reentered the hospital with a detachment below of some twenty days' duration; vision was 10/200. The visual field showed the vision limited to the lower part of the field. On April 1, operation was performed with poor results.

CASE 7.—F. D. M., aged 56, entered Mercedes Hospital on April 14, 1930, with a detachment of fifteen days' duration above and externally with limitation of the visual field below and internally.

The operation outlined was performed on April 21. Vision improved to 15/200. The visual field was the same.

## COMMENT

Given the small number and small range of cases in which this method has been tried and that in two of the cases a failure could be discounted no matter which treatment was used, any deduction as to either its proper or its comparative merits would be premature.

I have presented this modification so as to ask those who may consider my theoretical objections to Gonin's method as having some foundation to give this method a trial, without losing sight of the fact that if Gonin's statistics belie these theoretical objections, theories must give way to facts.

Of my seven cases, two were of traumatic origin; one seems to have been due to diabetes, three were secondary to cataract extractions and one was of unknown etiology. Three of the cases were failures, two being discountable before the operation; in one of the cases the success was most satisfactory, and in the other three some improvement was obtained or the process seems to have been brought to a standstill. I have had no cases due to high myopia.

I consider the traumatic cases as the most favorable. I also consider that it is useless to tackle cases that have lasted for some time. When the rupture can be localized, the prognosis is much more favorable. Youth also seems to be a beneficial factor. In reviewing the literature on the subject, I find that Addario [3] and Alajmo [4] in 1904 described a similar procedure. I have only seen them quoted by Uhthoff [5] and Lenz,[6] and have no knowledge of their technic or results, not having at hand their original papers.

---

3. Addario: Ann. d'ocul. **110**:36, 1893.

4. Alajmo: Ann. di ottal. e clin. ocul. **22**:542, 1893.

5. Uhthoff: Sammlung zwangloser Abhandl. a. d. Geb. d. Augenh., 1907, vol. 6, p. 8.

6. Lenz, in Graefe-Saemisch: Operationslehre, ed. 3, Berlin, Julius Springer, 1922, vol. 2, p. 1289.

# ATYPICAL (UPWARD) COLOBOMA OF THE CHOROID

## CLINICAL DESCRIPTION *

C. E. FINLAY, M.D.
HAVANA, CUBA

I have had the opportunity to observe the following case:

H. H. M., a woman, aged 27, came to the outpatient department of Mercedes Hospital on Jan. 18, 1930, complaining that for the last few months she had noticed that the sight of her left eye was poor. On examination the fundus of this eye was normal. That of the right eye showed an upward coloboma of the choroid. This (fig. 1) extended from the upper margin of the disk to the periphery of the fundus above, forming a sector somewhat over 30 degrees; it was pearl white, with pigment proliferation along its edges, especially below, where this encroached a little over the upper margin of the disk; a branched retinal vessel was to be seen coursing along its temporal margin, and at the lower inner angle some choroidal vessels were visible. There were no other retinal vessels in the patch. From its center an abnormal cilioretinal vessel coursed upward toward the periphery.

Skiascopic examination showed a hyperopia of +1.75 diopters in the right eye and a myopia of 0.50 diopter in the left eye. The sight of the right eye (with the coloboma) was normal, with a hypermetropia of +0.5. The left eye (with the normal fundus) had a visual acuity of 20/100, which was brought up to normal with a —1.50 sphere.

The visual field showed a characteristic sector-like defect downward, corresponding to the lesion in the right eye, in the upper part of which there was some light perception, the blind spot was somewhat enlarged (fig. 2). Familial antecedents were negative. The patient had five brothers and five sisters, all with normal eyes; each of her parents belonged to a family of six, none of whom complained of poor sight. Her maternal grandparents also had had normal eyes. The patient's mother had suffered from no pathologic disturbance or traumatism during the pregnancy prior to the patient's birth. There was no consanguinity between her parents.

### COMMENT

*Atypical Coloboma of the Choroid.*—Atypical coloboma of the choroid, not including under this denomination the so-called macular coloboma which often has a different origin (Mann [1]), is a rare lesion. I have found in the literature at my command seventeen cases; of these eight were in an upward direction and were reported by Lang,[2] Lindsay

* Submitted for publication, June 10, 1930.

1. Mann: Brit. J. Ophth. **11**:99, 1927.
2. Lang: Tr. Ophth. Soc. U. Kingdom **6**:489, 1886.

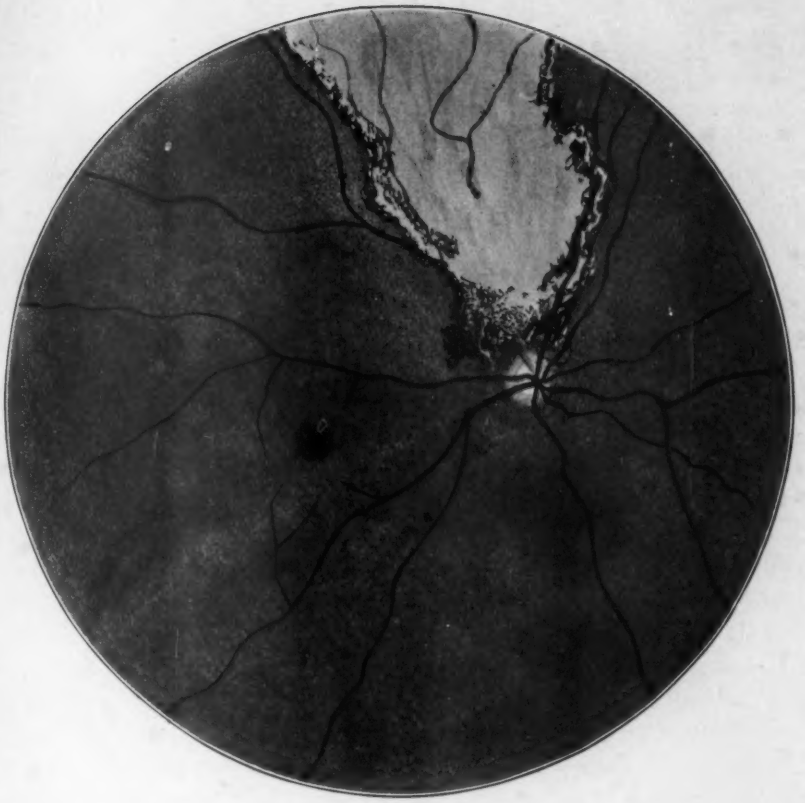


Fig. 1.—Coloboma of the right eye.

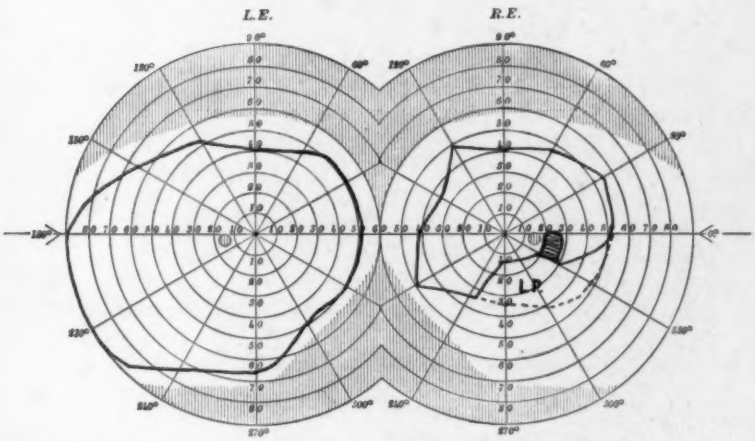



Fig. 2.—Visual field of the patient.

Johnson,[3] Rindfleisch,[4] Derby,[5] von Hippel,[6] Gifford,[7] Clausen,[8] and Mann and Ross;[9] none of them was as extensive as my case. The remaining nine were reported by Pflüger[10] (outward), Steinheim[11] (inward), Randall and de Schweinitz[12] (inward), Nuel and Le Plat[13] (outward), Frost[14] (outward), Pfanmüller[15] (outward), Raschowski[16] (outward), Parsons[17] (outward), Van Duyse[18] (down and inward).

The lesion varies in the different cases, both as regards its surface extent and as regards its depth, some cases showing a lesion limited to the choroid and outer layers of the retina—the retinal vessels coursing freely over the defect—while others include also the whole thickness of the retina (the two layers of the secondary optic vesicle), no retinal vessels being observable or only some proceeding abnormally from the ciliary system and constituting cilioretinal vessels, such as occurred in Mann and Ross'[9] case and in the one that I have just described.

*Pathogenesis.*—A delayed closure of the cleft of the secondary optic vesicle with secondary changes in the adjoining mesoderm, first advocated by Manz[19] and corroborated by Remāk, Schöler, Kölliker, von Hippel, Van Duyse and others, which so easily explains the production of typical coloboma, either by the interposition of mesodermic tissue or of abnormal vessels or by folding of the margins of the optic cup, was for a long time held not to explain that of atypical colobomas. However, the finding of supernumerary clefts in embryos of sheep,

3. Johnson, Lindsay: Arch. f. Augenh. **21**:290, 1890.

4. Rindfleisch, G.: Klin. Monatsbl. f. Augenh. **32**:91, 1894.

5. Derby, G. S.: Ophth. **3**:592, 1906-1907.

6. von Hippel, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1908, p. 1.

7. Gifford: Am. J. Ophth. **3**:99, 1920.

8. Clausen: Inaug. Dis., Tübingen, 1921.

9. Mann and Ross: Brit. J. Ophth **13**:12, 1929.

10. Pflüger: Arch. f. Augenh. **14**:1, 1884.

11. Steinheim: Centralbl. f. Augenh. **9**:201, 1886.

12. Randall and de Schweinitz: Arch. Ophth. **17**:454, 1888.

13. Nuel and Le Plat: Ann. d'ocul. **101**:151, 1889.

14. Frost: Tr. Ophth. Soc. U. Kingdom **13**:144, 1892-1893.

15. Pfanmüller, F.: Inaug. Diss., Giessen, 1894.

16. Raschowski, quoted by Peters: Angeborenen Fehler und Erkrankungen, Bonn, 1909, vol. 19, p. 22; quoted by Parsons: Pathology of the Eye, 1906, vol. 3, p. 1.

17. Parsons: Pathology of the Eye, New York, G. P. Putnams, 1906, vol. 3, p. 1.

18. Van Duyse: Arch. d'opht. **36**:22, 1919.

19. Manz, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 1, Leipzig, Wilhelm Engelmann, 1876, vol. 2, p. 58.

by von Ammon,[20] Van Duyse,[21] and Seefelder,[22] of the cow, by Van Duyse [21] and Seefelder; [22] of the pig, by Van Duyse,[21] Seefelder [22] and von Wolfrom; [23] of the rabbit, by von Wolfrom,[23] Seefelder [22] and Van Duyse, Jr.; [24] of the chick, by Meisner [25] and Seefelder [22] and, finally, in a human embryo by von Szily [26] prove that this explanation may also hold good for atypical colobomas, at least in some cases.

Deutschmann [27] considers a prior intra-uterine inflammation of the mesoderm necessary even for the production of typical colobomas. Elschnig [28] attributed the lesion to a glial hyperplasia; Bergmeister,[29] to choroidal hyperplasia, and Seefelder [30] to an embryonal aplasia; Mann [1] and Mann and Ross [9] maintained that in cases in which cilioretinal vessels are observed this implies a local destruction of Bruch's membrane which can be explained only by an inflammatory lesion.

It would seem that different elements may enter into the production of different cases. In the present case it would seem that the large extent of the lesion points to a nonclosure or a late closure of an accessory cleft which has involved both retinal layers of the optic cup, as proved by the absence of retinal vessels over the defect, while the presence of an abnormal cilioretinal vessel over its peripheral portion would seem to indicate a destructive lesion, at least at one point, with a local destruction of Bruch's membrane if one accepts Mann's and Mann and Ross' arguments in this respect, as I am inclined to do. The vessels belonging to the ciliary system develop at an earlier period of fetal life than do the normal retinal vessels.

---

20. von Ammon: Arch. f. Ophth. **4**:1, 1858.
21. Van Duyse: Arch. d'opht. **21**:94, 1901.
22. Seefelder: Algemeine Pathologie, 1914, p. 450.
23. von Wolfrom: Klin. Monatsbl. f. Augenh. **46**:227, 1908.
24. Van Duyse, G., Jr.: Brit. J. Ophth. **3**:529, 1919.
25. Meisner: Arch. d'opht. **79**:308, 1911.
26. von Szily: Klin. Monatsbl. f. Augenh. **45**:201, 1907.
27. Deutschmann: Klin. Monatsbl. f. Augenh. **19**:101, 1881.
28. Elschnig: Arch. f. Ophth. **51**:391, 1900.
29. Bergmeister: Arch. f. Ophth. **84**:1, 1913.
30. Seefelder: Tr. Fifth Congress, Heidelberg, 1911, p. 351; footnote 22.

# THE RÔLE OF LENS ANTIGEN AND UVEAL PIGMENT IN THE PRODUCTION OF HEREDITARY ANOMALIES OF THE EYE*

FREDERICK ALLISON DAVIS, M.D.
AND
HARVEY M. SMITH, PH.D.
MADISON, WIS.

In 1921, the attention of the senior author was directed to the work of Guyer and E. A. Smith in the experimental production of eye defects and particularly to the anatomic nature of the anomalies obtained. At that time a gross and microscopic study of the defective eyes of Dr. Guyer's stock was undertaken, the results of which were presented in a paper before the convention of English-speaking ophthalmologic societies in London, July, 1925.[1] No attempt was then made to analyze the results of Guyer and Smith, but the work was limited to a description of the lesions found. Briefly, this study showed that the defects obtained centered in the failure of the fetal ocular cleft to close or in its improper closure. The cleft remains open due to inhibition of growth of the developing optic vesicle or it is mechanically prevented from closing by ingrowth of mesodermal tissue. The former possibility appeared the more plausible. Typical coloboma, involving iris, ciliary body, choroid, retina and papilla was found, combined with varying grades of microphthalmus, staphyloma and cyst formation. The lens usually showed some abnormality, although at times it was normal when only the posterior portion of the cleft was involved. The lenses were at times completely absorbed, but usually showed varying degrees of opacity and deformation. They were more spherical than normal. These changes in the lens were apparently secondary to improper closure of the cleft.

In 1926, we undertook a series of injections with the hope of reproducing the lesions that had been obtained in the injection experiments of Guyer and Smith. The present report is based on the experiments carried on during the past three years. Although we have been dis-

* Submitted for publication, June 6, 1930.

* From the Department of Ophthalmology, University of Wisconsin.

* This paper was presented before the Thirteenth International Congress of Ophthalmology, held in Amsterdam in 1929. A brief abstract of the paper appears in "The Transactions," but permission was received to publish the complete paper in the ARCHIVES.

1. Davis, F. A.: Hereditary Eye Defects in Rabbits Experimentally Induced, Tr. Ophth. Soc. U. Kingdom **45**:555, 1925.

appointed in our failure to reproduce the defects obtained by Guyer and Smith, we feel that the number of experiments performed justifies a report at this time.

## DESCRIPTION OF EXPERIMENTS

When these experiments were planned we deliberately chose the second method by which Guyer and Smith had obtained positive results, namely, the direct immunization of rabbits with lens substance. This method was chosen because of its obviously greater practical interest in connection with lesions of the human eye, particularly in connection with the problem of traumatic cataract and the possible effect of needled lenses. In addition, a few lenses of pregnant mothers were needled and some injections of uveal pigment were made, the latter because of the relation of uvea to coloboma, and the fact that the defects anticipated are all related to coloboma. Further, the experiments of Woods [2] concerning the treatment for sympathetic ophthalmia indicate that specific proteins are present in this pigment. The injections were performed as nearly as possible by the same technic as that used by Guyer and Smith, a fact that is not true of the work of some other investigators who have attempted to repeat this work.

We intentionally avoided using any local stock that might possibly have been contaminated by some of Dr. Guyer's hereditary strains, but secured entirely unrelated stock from a breeder in Oshkosh, Wis. The experiments were carried on entirely independent of Dr. Guyer's work, the breeding pens being quartered in the University of Wisconsin Medical School.

Aseptic technic was followed in the preparation and injection of the lens protein solution. The cornea and conjunctiva were sterilized, the cornea removed, the lens capsule ruptured and the lens delivered and washed in physiologic solution of sodium chloride. Thus we were certain of dealing with lens protein only and not with vitreous and aqueous as well. The fresh lenses were ground in a mortar with salt solution and centrifugated to remove the coarser particles.

The dosage of rabbit lens was always in terms of the number of lenses used, the solution being diluted with salt solution to yield a convenient dosage (from 3 to 5 cc.) per subject. Beef lens dosage was in terms of the number of cubic centimeters of a standard solution, usually four lenses to 30 cc. of salt solution, from 1 to 4 cc. being injected. In view of the work of Uhlenhuth, Hektoen and others on the specificity of lens protein it seems to us immaterial whether rabbit or beef lens is used, although the former was always chosen if available, since it was used in Guyer and Smith's successful injections.

---

2. Woods, A. C.: Sympathetic Ophthalmia: The Use of Uveal Pigment in Diagnosis and Treatment, Tr. Ophth. Soc. U. Kingdom **45**:419, 1925.

TABLE 1.—*Lens Injections in Attempts to Produce Eye Defects in Offspring of Injected Mothers*

| Experiment No. | Female and Male | Date of Injection | Days of Pregnancy | Amount and Nature of Injection* | Results |
|---|---|---|---|---|---|
| I | ♀ 03 | 10/ 5/26 | — 5 | P. 3 young R. lenses | 5 young; born 11/11/26; |
| | ♂ 326 | 10/ 7/26 | — 3 | P. 4 young R. lenses | all normal |
| | | 10/12/26 | + 2 | P. 4 young R. lenses | |
| II | ♀ 03 | 1/11/27 | + 3 | P. 5 cc., 4:30 B. lens | 5 young; born 2/9/27; |
| | ♂ S2 | 1/13/27 | + 5 | P. 5 cc., 4:30 B. lens | all normal |
| | | 1/18/27 | +10 | P. 5 cc., 4:30 B. lens | |
| III | ♀ 03 | 4/ 7/27 | + 2 | I. 1 cc., 3:30 B. lens | 5 young; born 5/7/27; |
| | ♂ 012 | 4/ 9/27 | + 4 | I. 1 cc., 3:30 B. lens | all normal |
| | | 4/11/27 | + 6 | Left lens needled | |
| | | 4/27/27 | +22 | Second needling of left lens | |
| IV | ♀ 04 | 10/ 7/26 | .... | P. 5 young R. lenses | Would not mate |
| | ♂ None | 10/12/26 | .... | P. 4 young R. lenses | |
| | | 10/14/26 | .... | P. 4 young R. lenses | |
| V | ♀ 05 | 10/27/26 | +15 | P. 4 young R. lenses | 6 young; born 11/12/26; |
| | ♂ 326 | 11/ 4/26 | +23 | P. 2 young R. lenses | all normal |
| VI | ♀ 05 | 12/29/26 | + 1 | S. 1 cc., 4:30 B. lens | 5 young; born 1/30/27; |
| | ♂ 012 | 12/30/26 | + 2 | S. 1 cc., 4:30 B. lens | all normal |
| | | 12/31/26 | + 3 | P. 3 cc., 4:30 B. lens | |
| | | 1/ 4/27 | + 7 | P. 4 cc., 4:30 B. lens | |
| VII | ♀ 05 | 3/23/27 | —34 | I. 1 cc., 4:30 B. lens | 6 young; born 5/28/27; |
| | ♂ 012 | 3/28/27 | —29 | I. 1 cc., 4:30 B. lens | all normal |
| | | 3/31/27 | —26 | I. 1.5 cc., 3:25 B. lens | |
| VIII | ♀ 05 | 12/28/27 | .... | No further injections | 7 young; born 11/28/27; |
| | ♂ 019A5 | | | | 4 living, normal |
| IX | ♀ 05 | 4/18/28 | + 8 | I. 2 R. lenses | 6 young; born 5/11/28; |
| | ♂ 019A5 | | | | all normal |
| X | ♀ 08 | 10/19/26 | + 2 | P. 4 young R. lenses | Young apparently died in |
| | ♂ 326 | 10/21/26 | + 4 | P. 4 young R. lenses | the uterus, not examined |
| | | 10/26/26 | + 9 | P. 4 young R. lenses | |
| XI | ♀ 08 | 12/10/26 | 0 | P. 1 cc., 4:6 R. lens | Did not produce young |
| | ♂ S2 | 12/11/26 | + 1 | P. 2 cc., 4:6 R. lens | |
| | | 12/14/26 | + 4 | P. 4 cc., 4:20 B. lens | |
| | | 12/16/26 | + 6 | P. 3 cc., 4:30 B. lens | |
| XII | ♀ 08 | 2/ 2/27 | — 3 | I. 1 cc., P. 4 cc., 4:30 B. lens | Operation at 28 days; |
| | ♂ S2 | 2/ 5/27 | 0 | I. 2.5-3 cc., 4:30 B. lens | not pregnant |
| | | 2/ 9/27 | + 4 | I. 3 cc., 4:30 B. lens | |
| | | 2/11/27 | + 6 | I. 3 cc., 4:30 B. lens | |
| XIII | ♀ 011 | 12/10/26 | +11 | P. 2 adult R. lenses | Abortion at 24 days; |
| | ♂ 012 | 12/14/26 | +15 | P. 4 cc., 4:20 B. lens | embryos chewed; no |
| | | 12/16/26 | +17 | P. 3 cc., 4:20 B. lens | autopsy possible |
| XIV | ♀ 011 | 1/11/27 | .... | P. 5 cc., 4:30 B. lens | Died in shock after sec- |
| | ♂ None | 1/13/27 | .... | I. 2 cc., 4:30 B. lens | ond injection |
| XV | ♀ 015 | 12/14/26 | — 3 | P. 4 cc., 4:20 B. lens | 5 young; 2 died at birth; |
| | ♂ S2 | 12/16/26 | — 1 | P. 3.5 cc., 4:20 B. lens | 3 normal |
| | | 12/21/26 | + 4 | P. 4 cc., 4:20 B. lens | |
| XVI | ♀ 018A4 | 10/29/27 | +10 | I. 1 R. lens; previous in- | 8 young; all normal |
| | ♂ 019A4 | | | jection; I. R. pigment | |
| XVII | ♀ 018A4 | 3/19/28 | + 5 | I. 1 R. lens | 9 young; born 4/15/28; |
| | ♂ 515A4 | | | | all normal |
| XVIII | ♀ 020A3 | 2/17/28 | + 8 | I. 2 cc.; 5:25 B. lens | 7 young; born 3/12/28; |
| | ♂ 019A5 | 2/20/28 | +11 | I. 2 cc., 5:25 B. lens | all normal |
| | | 2/23/28 | +14 | I. 3 cc., 5:25 B. lens | |
| | | 2/27/28 | +18 | I. 2 R. lenses | |
| XIX | ♀ 022A2 | 10/24/27 | + 2 | I. 1 R. lens | 7 young; born 5/22/28; |
| | ♂ 019A5 | 10/26/27 | + 4 | I. 1 R. lens | all normal |
| | | 10/29/27 | + 7 | I. 1 R. lens | |
| XX | ♀ 022A2 | 2/29/28 | + 9 | I. 2 cc., 5:25 B. lens | 6 young; born 3/23/28; |
| | ♂ 019A4 | | | Subsequent pig. inj. q.v. | all normal |
| XXI | ♀ 023C | 2/29/28 | .... | I. 2 R. lenses | Not pregnant |
| | ♂ 023A,B | 3/ 2/28 | .... | I. 1 R. lens | |
| | | 3/ 6/28 | .... | I. 2 R. lenses | |

* P. indicates peritoneal; I., intravenous; R., rabbit; B., beef.

TABLE 1.—*Lens Injections in Attempts to Produce Eye Defects in Offspring of Injected Mothers—Continued*

| Experiment No. | Female and Male | Date of Injection | Days of Pregnancy | Amount and Nature of Injection* | Results |
|---|---|---|---|---|---|
| XXII | ♀ 024B | 3/ 6/28 | + 6 | I. 2 R. lenses | 7 young; born 3/31/28; all normal |
| | ♂ C3A | 3/15/28 | +15 | I. 2 R. lenses | |
| XXIII | ♀ C1 | 10/14/27 | + 7 | I. ½ R. lens | 6 young; born 11/27/26; all normal |
| | ♂ 019A4 | 10/15/27 | + 8 | I. ½ R. lens | |
| | | 10/17/27 | +10 | I. ⅝ R. lens | |
| XXIV | ♀ C1 | 3/19/28 | + 5 | I. 1 R. lens | 7 young; born 4/14/28; all normal |
| | ♂ C3A | | | | |
| XXV | ♀ C2A1 | 2/17/27 | + 8 | I. 2 cc., 5:25 B. lens | Did not produce young |
| | ♂ 019A5 | 2/20/28 | +10 | I. 2 cc., 5:25 B. lens | |
| | | 2/23/28 | +13 | I. 3 cc., 5:25 B. lens | |
| | | 2/27/28 | +17 | I. 3.5 cc. (slight shock) | |
| XXVI | ♀ C2A2 | 2/29/28 | + 9 | I. 2 cc., 5:25 B. lens | 8 young; born 3/22/28; all normal |
| | ♂ 022A4 | 3/ 2/28 | +11 | I. 1 R. lens | |
| | | 3/ 6/28 | +15 | I. 2 R. lenses | |
| XXVII | ♀ C1 | 5/23/28 | .... | I. ⅜ R. lens | Died several days after last injection |
| | | 5/30/28 | .... | I. 2 R. lenses | |
| XXVIII | ♀ 020A3 | 5/23/28 | −52 | I. ¾ R. lens | 2 young; born 8/14/28; all normal |
| | ♂ C3A | 5/30/28 | −45 | I. 2 R. lenses | |
| | | 6/ 8/28 | −36 | I. 2 R. lenses | |
| | | 6/21/28 | −23 | I. 2 R. lenses | |
| | | 6/26/28 | −18 | I. 1.7 R. lenses | |
| | | 7/ 2/28 | −12 | I. 1.4 R. lenses | |
| | | 7/16/28 | + 2 | I. 0.5 pig. lens | |
| | | 7/21/28 | + 7 | I. 2 R. lenses | |
| XXIX | ♀ 025A | 5/23/28 | −52 | I. ¾ R. lens | 8 young; born 8/14/28; all normal |
| | ♂ C3A | 5/30/28 | −45 | I. 2 R. lenses | |
| | | 6/ 8/28 | −36 | I. 2 R. lenses | |
| | | 6/26/28 | −18 | I. 1.7 R. lenses | |
| | | 7/ 2/28 | −12 | I. 1.4 R. lenses | |
| | | 7/16/28 | + 2 | I. 0.5 pig. lens | |
| | | 7/21/28 | + 7 | I. 2 R. lenses | |
| XXX | ♀ 221A3 | 5/23/28 | .... | I. ¾ R. lens | Died 7/13/28; pneumonia |
| | ♂ None | 5/30/28 | .... | I. 2 R. lenses | |
| | | 6/ 8/28 | .... | I. 2 R. lenses | |
| | | 6/21/28 | .... | I. 2 R. lenses | |
| | | 6/26/29 | .... | I. 1.7 R. lenses | |
| | | 7/ 2/29 | .... | I. 1.4 R. lenses | |
| XXXI | ♀ 024A | 5/30/28 | .... | I. 2 R. lenses | Died 7/13/28; emaciated |
| | | 6/ 8/28 | .... | I. 2 R. lenses | |
| | | 6/21/28 | .... | I. 2 R. lenses | |
| | | 6/26/28 | .... | I. 1.7 R. lenses | |
| | | 7/ 2/28 | .... | I. 1.4 R. lenses | |
| XXXII | ♀ 024B | 5/30/28 | .... | I. 2 R. lenses | Died 6/28/28; emaciated |
| | | 6/ 8/28 | .... | I. 2 R. lenses | |
| | | 6/21/28 | .... | I. 2 R. lenses | |
| | | 6/26/28 | .... | I. 1.7 R. lenses | |
| XXXIII | ♀ 026A | 5/30/28 | .... | I. 2 R. lenses | Would not mate |
| | | 6/21/28 | .... | I. 2 R. lenses | |
| | | 6/26/28 | .... | I. 1.7 R. lenses | |
| | | 7/ 2/28 | .... | I. 1.4 R. lenses | |
| | | 7/16/28 | .... | I. 0.5 pig. lenses | |
| XXXIV | ♀ 026B | 5/30/28 | .... | I. 2 R. lenses | Killed by mate 026A 6/4/28 |
| XXXV | ♀ 019A1 | 6/26/28 | −17 | I. 1.7 R. lenses | 5 young; born 8/13/28; all normal |
| | ♂ 017A5 | 7/ 2/28 | −11 | I. 1.4 R. lenses | |
| | | 7/16/28 | + 3 | I. 0.5 pig. lenses | |
| | | 7/20/28 | + 7 | I. 2 R. lenses | |
| XXXVI | ♀ 177D2 | 12/20/28 | −6 mo. | I. 1 R. lens | 7 young; born 7/20/29; all normal |
| | ♂ 019A5 | 12/29/28 | −171 | I. 1½ R. lenses | |
| | | 4/13/29 | −66 | I. 1 R. lens | |
| | | 4/27/29 | −53 | I. 1 R. lens | |
| | | 5/11/29 | −39 | I. 1.5 R. lenses | |
| XXXVII | ♀ 177D3 | 12/20/28 | −146 | I. 1 R. lens | 7 young; born 6/16/29; all normal |
| | ♂ 019A4 | 12/29/28 | −137 | I. 1½ R. lenses | |
| | | 4/13/29 | −32 | I. 1 R. lens | |
| | | 4/27/29 | −18 | I. 1 R. lens | |
| | | 5/11/29 | − 4 | I. 1.5 R. lenses | |

* P. indicates peritoneal; I., intravenous; R., rabbit; B., beef.

TABLE 1.—*Lens Injections in Attempts to Produce Eye Defects in Offspring of Injected Mothers—Continued*

| Experiment No. | Female and Male | Date of Injection | Days of Pregnancy | Amount and Nature of Injection* | Results |
|---|---|---|---|---|---|
| XXXVIII | ♀ 177D4 | 12/20/28 | —154 | I. 1 R. lens | 6 young; born 6/28/29; all normal |
| | ♂ 019A5 | 12/29/28 | —145 | I. 1½ R. lenses | |
| | | 4/13/29 | —40 | I. 1 R. lens | |
| | | 4/27/29 | —26 | I. 1 R. lens | |
| | | 5/11/29 | —12 | I. 1.5 R. lenses | |
| XXXIX | ♀ 177D1 | 12/20/28 | —154 | I. 1 R. lens | 3 young; born 6/28/29; all normal |
| | ♂ S19D | 12/29/28 | —145 | I. 1.3 R. lenses | |
| | | 4/13/29 | —40 | I. 1 R. lens | |
| | | 4/27/29 | —26 | I. 1 R. lens | |
| | | 5/11/29 | —12 | I. 1.5 R. lenses | |
| XL | ♀ S17A | 12/20/28 | —154 | I. 1 R. lens | 2 died, normal; 5 left, not studied |
| | ♂ S15A5 | 12/29/28 | —145 | I. 1½ R. lenses | |
| | (known | 6/14/29 | — 9 | I. 1 R. lens | |
| | defective | 6/18/29 | — 5 | I. 1.5 R. lenses | |
| | strain) | 6/21/29 | — 2 | I. 2 R. lenses | |
| | | 6/28/29 | + 5 | I. 3 R. lenses | |
| XLI | ♀ S17B | 12/20/28 | —5 mo. | I. 1 R. lens | 6 young; 1 abnormal; 2 died, normal; 3 left, not studied |
| | ♂ S15A4 | 12/29/28 | —5 mo. | I. 1½ R. lenses | |
| | (known | 6/14/29 | —15 | I. 1.5 R. lenses | |
| | defective | 6/18/29 | —11 | I. 2 R. lenses | |
| | strain) | 6/21/29 | — 8 | I. 3 R. lenses | |
| | | 6/28/29 | — 1 | I. 3 R. lenses | |
| XLII | ♀ S17E | 12/20/28 | —5 mo. | I. 1 R. lens | No young |
| | ♂ S15A4 | 12/29/28 | —5 mo. | I. 1½ R. lenses | |
| | (known | 6/14/29 | —11 | I. 1 R. lens | |
| | defective | 6/18/29 | — 7 | I. 1.5 R. lenses | |
| | strain) | 6/21/29 | — 4 | I. 2 R. lenses | |
| | | 6/28/29 | + 3 | I. 3 R. lenses | |
| XLIII | ♀ S15A2 | 6/14/29 | —15 | I. 1 R. lens | No young |
| | ♂ S15A5 | 6/18/29 | —11 | I. 1.5 R. lenses | |
| | (known | 6/21/29 | — 8 | I. 2 R. lenses | |
| | defective | 6/28/29 | — 1 | I. 3 R. lenses | |
| | strain) | | | | |
| XLIV | ♀ S18A | 6/14/29 | —15 | I. 1 R. lens | No young |
| | ♂ S15A4 | 6/18/29 | —11 | I. 1.5 R. lenses | |
| | (known | 6/21/29 | — 8 | I. 2 R. lenses | |
| | defective | 6/28/29 | — 1 | I. 3 R. lenses | |
| | strain) | | | | |

* P. indicates peritoneal; I., intravenous; R., rabbit; B., beef.

TABLE 2.—*Primary Lens Needling*

| Experiment No. | Female and Male | Date of Injection | Days of Pregnancy | Details of Treatment How Needled | Results |
|---|---|---|---|---|---|
| XLV | ♀ 03 | 4/11/27 | + 6 | Left lens needled; previous lens injection | 5 young; all normal |
| | ♂ 012 | 4/27/27 | —22 | Left lens needled | |
| XLVI | ♀ 05 | 4/27/27 | + 1 | Left lens; previous lens injection | 6 young; all normal |
| | ♂ 012 | | | | |
| XLVII | ♀ 019A3 | 2/29/28 | + 9 | Left lens; previous needling right lens and iris | 8 young; normal |
| | ♂ 022A4 | 3/ 6/28 | —15 | Both lenses needled | |
| XLVIII | ♀ 023D | 2/29/28 | .... | Both lenses needled | 5 young; first mating no litter; second mating, same male, 5 young; all normal |
| | ♂ 023A,B | 3/ 6/28 | .... | Right lens needled | |
| XLIX | ♀ 024A | 3/ 6/28 | + 4 | Needling both lenses | 7 young; all normal |
| | ♂ C3A | 3/15/28 | —13 | Needling both lenses | |

Lens needling was performed by means of a Ziegler knife needle; in each case we cut repeatedly into the substance of the lens. Rabbit iris pigment was prepared, under aseptic precautions, by removing whole irises, grinding in a mortar, allowing the coarser sediment to settle out or filter through gauze. When beef pigment was used, the whole uveal tract was dissected out and treated in the foregoing manner. Some of the earlier injections were made intraperitoneally, but all the later ones were done intravenously, the marginal vein of the ear being used.

TABLE 3.—*Pigment Injections for Defective Young*

| Experiment No. | Female and Male | Date of Injection | Days of Pregnancy | Amount and Nature of Injection* | Results |
|---|---|---|---|---|---|
| L | ♀ 07 | 12/29/26 | — 5 | P. 3 cc. 4:30 B. uvea | 6 young; all normal |
| | ♂ 012 | 12/31/26 | — 3 | P. 3 cc. 4:30 B. uvea | |
| | | 1/ 4/27 | 1 | P. 3 cc. 4:30 B. uvea | |
| LI | ♀ 07 | 4/ 7/27 | 2 | I. 1 cc. 4:40 B. uvea | Died in anaphylaxis |
| | ♂ 012 | | | | |
| LII | ♀ 09 | 10/19/26 | —15 | P. 2.5 cc. 4:4 R. iris | 6 young; 1 with displaced opaque lens, keratitis, hydrophthalmos |
| | ♂ 326 | 10/21/26 | —11 | P. 2.5 cc. 4:4 R. iris | |
| | | 10/26/26 | — 6 | P. 2 cc. 4:4 R. iris | |
| LIII | ♀ 09 | 2/ 2/27 | — 3 | I. 2 cc. 4:30 B. uvea | Died in convulsions after second injection |
| | ♂ 012 | 2/ 5/27 | 0 | I. 4 cc. 4:30 B. uvea | |
| LIV | ♀ 018A3 | 10/24/27 | —29 | I. 1 R. uvea | 7 young; 3 died at birth; 4 normal |
| | ♂ 022A4 | 10/26/27 | —27 | I. 1 R. uvea | |
| LV | ♀ 018A4 | 10/26/27 | — 7 | I. 1 R. uvea | 8 young; all normal |
| | ♂ 019A4 | 10/29/27 | | Lens injection | |
| LVI | ♀ 85 | 12/14/26 | — 4 | P. 2 cc. 4:20 B. uvea | 7 young; 2 died at birth; 5 normal |
| | ♂ 82 | 12/16/26 | — 2 | P. 3 cc. 4:20 B. uvea | |
| | | 12/18/26 | 0 | P. 3 cc. 4:20 B. uvea | |
| LVII | ♀ 85 | 2/28/27 | 3 | I. 1 cc. 3:30 B. uvea | Died in shock after last injection |
| | ♂ 82 | 3/ 2/27 | 5 | I. 1.5 cc. 3:30 B. uvea | |
| | | 3/ 4/27 | 7 | I. 1.8 cc. 4:30 B. uvea | |
| LVIII | ♀ 022A2 | 3/ 2/28 | 11 | I. 2 R. irises | 6 young; normal |
| | ♂ 019A4 | 3/ 6/28 | 15 | I. 2 R. irises | |
| LIX | ♀ 022A5 | 2/22/28 | 9 | I. 2 R. irises | 7 young; normal |
| | ♂ 019A5 | 2/25/28 | 12 | I. 2 R. irises | |
| | | 2/29/28 | 16 | I. 2 R. irises | |
| LX | ♀ 019A1 | 3/19/28 | 6 | I. 2 R. irises | 8 young; 1 corneal opacity; conjunctivitis |
| | ♂ 022A4 | | | | |
| LXI | ♀ 023D | 4/18/28 | —30 | 2 R. irises | 5 young; born 6/24/29; all normal |
| | ♂ 022A4 | 5/ 8/28 | —16 | 1 R. iris | |

* P. indicates peritoneal; I., intravenous; R., rabbit; B., beef.

Table 1 records the earlier experiments, their timing in relation to pregnancy, their details and results. In all, forty-four series of injections into mated females were made with twenty-seven pregnancies coming to term, yielding one hundred and ninety-two normal young. No defective eyes were obtained in this series. Experiments 28 to 35 require special mention in that they represent an attempt to repeat Guyer and Smith's successful experiment with their series 84, both in dosage of lens and in timing in relation to pregnancy, and also in the fact that injections were made into the male as well as into the female.

Table 2 records a small series of experiments on the needling of the lens of the pregnant mother, twenty-three normal young being obtained from this series.

Table 3 records a series of injections of uveal pigment into pregnant rabbits. In all, twelve experiments were performed, the matings producing a total of sixty young. Among these there was one with an abnormal eye. One of the young of female 09, experiment 52, showed on first examination at 20 days a diffuse haziness and subluxation of the lens. The eye later went through a series of interesting changes. The cornea became diffusely cloudy due to infiltration of the stroma. This

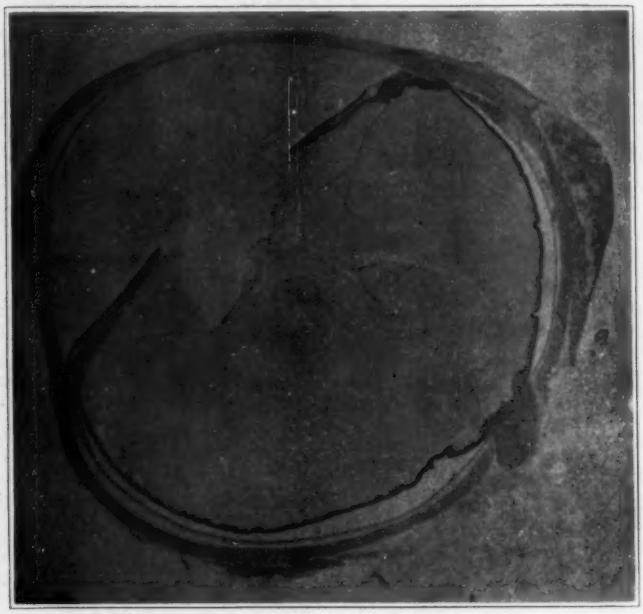


Fig. 1.—Section of the globe of an animal obtained in experiment 52. The eye shows an early stage of hydrophthalmus with secondary corneal changes. The filtration angle is practically absent and the characteristic changes are noted in the choroid. See text.

was accompanied by a gradual enlargement and moderate staphyloma of the entire cornea, with increase in intra-ocular tension. A central ulceration of the cornea developed. Clinically, the eye gave the appearance of an early hydrophthalmos with secondary involvement of the cornea. The clinical diagnosis was confirmed when the eye was later sectioned. (Killing of the animal was necessary because of increasing general weakness and paralysis of the hind legs.)

Pathologic examination of this eye (figs. 1 and 2) revealed changes fairly typical of an early hydrophthalmos with the afore-

mentioned corneal involvement. The filtration angle of the eye is of particular interest. In spite of the fact that the anterior chamber was very deep, the base of the iris was attached directly to the cornea, and the iris pillars and the large spaces of the uveal meshwork were absent. Schlemm's canal was present in some of the sections, though absent in others, and when present was reduced in size. This condition represents an arrest in development, former studies indicating that the meshwork of this angle develops after birth.[3] The cornea was thin and showed interstitial keratitis, a large central area of ulceration with con-

Fig. 2.—A higher magnification of the filtration angle of the section illustrated in figure 1. Note the complete absence of the filtration angle which is so highly developed in the normal rabbit eye. (Figs. 3 and 4 are inserted to direct attention to the normal filtration angle of the eye of the rabbit.)

siderable scar formation. At one place Descemet's membrane only remained, the other layers having been destroyed. Adjacent to this area the cornea was thickened, the stroma being replaced by scar tissue. Under this area Descemet's membrane was absent, probably due to rupture from tension. The sclera varied little from the normal in

3. Davis, F. A.: The Anatomy and Histology of the Eye and Orbit of the Rabbit, Tr. Am. Ophth. Soc. **27**:401, 1929.

Fig. 3.—Filtration angle of the normal albino rabbit. *1* indicates the cornea; *2*, the sclera; *3*, Descemet's membrane; *4*, Descemet's endothelium; *5*, Schlemm's canal; *6*, corneoscleral trabeculum; *7*, uveal meshwork; *8*, iris pillars; *9*, Descemet's membrane splitting to clothe partially a pillar; *10*, iris; *11*, veins. Van Gieson stain; × 108. (Compare with fig. 2).

Fig. 4.—Filtration angle of the eye of the normal albino rabbit; higher magnification of figure 3.

thickness. The iris and ciliary body showed an acute inflammatory reaction with pus cells being discharged into the anterior and posterior chambers and the vitreous. The retina was fairly normal, but the choroid showed much extravasated blood and engorgement of the veins. The vitreous was more densely fibrous than normal, resembling the embryonic condition. Figures 3 and 4 are presented to direct attention to the normal filtration angle of the eye of the rabbit. The lens was displaced downward, so that its edge was near the middle of the pupil. It was also displaced somewhat backward, thus contributing to the depth of the chamber. The nucleus showed degeneration of its fibers. We regard this eye as a case of developmental anomaly of unknown origin. The brothers and sisters of this animal have been used repeatedly in subsequent experiments, and their offspring have always been normal.

The eyes of all the treated animals as well as those of the offspring were examined with focal illumination and the loupe, supplemented by ophthalmoscopic examination in many instances. Microscopic study of the eyes was not done as a routine measure, since they appeared normal, but occasionally they were sectioned to be used in connection with other studies. Many of the offspring of the animals from all three series have been mated in subsequent work without the appearance of a defective eye. In some cases (experiments 18, 19, 20, 21, 22, 25, 26, 28, 29, 31, 32, 33, 34 and 35) the female used was the daughter of a treated mother. Our results, therefore, combined with the evidence of a number of other investigators whose work will be referred to in detail later, lead us to the conclusion that anomalies such as were described by Guyer and Smith are not due to the effect of injected lens substance, but are more likely hereditary defects of unknown origin. Since our work has led us to conclusions that are at variance with the results of Guyer and Smith, and since other investigators have also attempted to repeat their work, an analysis of their observations seems necessary.

## SUMMARY AND ANALYSIS OF OBSERVATIONS OF GUYER AND SMITH

Guyer and E. A. Smith, in a series of papers published in 1918, 1920 and 1924, reported that they induced lesions of the eye in three entirely separate and unrelated strains of rabbits, which in each instance have become hereditary, by the treatment of pregnant rabbits with lens sensitized fowl serum and the direct injection of lens substance. They present a fairly convincing array of evidence for their deductions, and we shall set forth a few of these facts.

The first animal from which they obtained defective-eye offspring, female B, had been previously bred, but the litter showed no eye defects. The male is not identified. After treatment with lens sensitized fowl serum, a litter was born in which one with a defective eye appeared,

namely, 3A1. Subsequent breeding of this female, B, after a second series of treatments resulted in a litter of six, all of which had normal eyes. We studied the eye of 3A1 histologically in detail. It showed an extreme example of microphthalmus with cyst, which so often complicates coloboma. The lens was round and calcified, filling the entire vitreous cavity. It is our opinion that the condition of the lens is secondary to the coloboma and is not the result of the treatment. This was discussed in detail by the senior author in a previous communication.[1]

In their 1920 paper, Guyer and Smith[4] published a genealogical tree showing the results of breeding male 3A1 with his sister and inbreeding the offspring. The genealogical tree demonstrates conclusively that the condition is hereditary, and, further, that it is not a pure mendelian recessive.

The chief criticism of the experiment in which male 3A1 was obtained (experiment 1, 1918[5]) is that the ancestry of neither of the parents is stated. Therefore, since spontaneous appearance of hereditary coloboma with microphthalmus is well known in the rabbit, there is a possibility that this occurred in the case of male 3A1. In this connection it is an interesting fact that female 22[6] was mated to male 2 (the father of 3A1) and treated. The offspring were normal, but an animal of the third generation, obtained by inbreeding the offspring, was defective. It seems, therefore, that male 2 may have been a recessive carrier of coloboma.

Guyer and Smith recorded in their 1920 paper[6] the development of a second defective strain, namely, the "16A1" line. Defective eyes were presumably induced in the offspring of female 16A1 by the injection of lens sensitized fowl serum. Briefly, this series of experiments revealed that the first treatment of 16A1, after mating with her brother, 16A2, resulted in a normal litter. A second experiment, conducted three months later, which consisted of injections on the twelfth and fourteenth days of pregnancy and again mating with the same brother, yielded one defective-eyed young among a litter of four. A third treatment and mating with a foreign male (50) yielded two colobomatous young of a litter of five. While these results are consistent with the known facts regarding hereditary coloboma, according to von Szily, they might be used to support the hypothesis of the specific effect of the treatment, since its action might logically be considered to be greater in the second and third matings than in the first. Serious consideration should be given the control experiments with related animals; namely, the sisters

4. Guyer, M. F., and Smith, E. A.: Studies on Cytolysins: II. Transmission of Induced Eye Defects, J. Exper. Zool. **31**:171, 1920.

5. Guyer, M. F., and Smith, E. A.: Studies on Cytolysins: I. Some Prenatal Effects on Lens Antibodies, J. Exper. Zool. **26**:65, 1918.

6. Guyer and Smith (footnote 4, p. 197).

of 16A1—the second female which produced the defective-eyed young. Several of these were mated to male 16A2 and male 50, the males used in the successful experiment, but no eye lesions were observed in the offspring. Further, and more convincing, is the statement that the normal-eyed young (N16 series) of female 16A1 and her brother, 16A2, obtained after the first series of treatments were interbred and every young of 18 resulting had normal eyes. Guyer and Smith felt that if a hereditary strain had been present, inbreeding of the offspring of these animals would likely have brought out a defect. Here, again, the ancestry of the 16A family is unknown, as they were "born from a female shipped from Minneapolis, pregnant when she arrived at the laboratory." The second mate of 16A1, male 50, also of unknown ancestry, was shipped from Chicago. The possibility of a hereditary taint cannot here be denied.

A third defective strain was obtained by Guyer and Smith[7] from a rabbit (female 84) that had received injections directly with pulped rabbit lens. Her mate had likewise been given an injection. She was "the daughter of an albino doe which came in a shipment from Lafayette, Indiana." Here, again, the ancestry is unknown. The first treatment (and mating with male 25) yielded normal young. A second mating with male 4A7, a member of the 3A1 defective strain, yielded one defective animal. A third mating, with male 62, "of a different and normal-eyed strain," accompanied by treatment of both parents, yielded one defective-eyed young (92B1) of a litter of five. This eye was also described by one of us (F. A. D.[8]) in 1925. A number of animals received the same treatment as female 84[8] but no defective eyes were obtained, while female 84 produced several defective-eyed young with different matings. It thus seems possible again that the authors were unfortunate in obtaining in female 84 a recessive carrier of coloboma, since the results obtained in these experiments are explainable by the known behavior of hereditary coloboma. Guyer and Smith pointed out that intensive inbreeding of the brother and sisters of female 84 produced no defective young, indicating that there was no recessive defect in the strain. This, of course, lends weight to their argument but it is not conclusive, since it is possible, on the basis of the laws of hereditary, that one individual of a family may have a recessive character, the others being free from it. It must be admitted that it is a remarkable fact that these authors should accidentally have happened onto three separate defective strains in the course of their experiments, and this lends considerable weight to their argument. It is also noteworthy that numerous

7. Guyer and Smith: Further Studies on Inheritance of Eye Defects Induced in Rabbits, J. Exper. Zool. **38**:449, 1924.

8. Guyer and Smith (footnote 7, table 4, p. 456).

9. Guyer and Smith (footnote 7, figure II, p. 458).

control experiments, in which normal chicken serum and other foreign proteins were used, all produced negative results.

Von Szily [10] made an exhaustive criticism of the work of Guyer and Smith, based entirely on their two earlier papers, and came to the conclusion that their results are entirely accidental, and that they are dealing with simple hereditary coloboma. He attempted to show that male 2, the father of 3A1, was also the father of the 16A1 strain. This conclusion is unjustified on the basis of the evidence in the papers reviewed, and from Guyer and Smith's 1924 paper (which von Szily had not seen) is found to be untrue. Male 50, obtained from Chicago, was the father of this strain. Von Szily seriously questioned this experiment, due to the fact that he thought that only a single injection had been given prior to the birth of the defective-eyed animal. He failed to note that this mother, female 16A1, had previously received a series of injections about two months prior to the mating which resulted in this defective animal. He pointed out the ignorance of Guyer and Smith regarding the inheritance of coloboma and its spontaneous occurrence in rabbits, which is not surprising in view of the fact that they are biologists and are unfamiliar with ophthalmologic literature. Von Szily further stated that the inbreeding experiments with male 3A1 show the behavior of typical hereditary coloboma. He also said that it would be necessary to use animals that had shown no eye defects after three generations of inbreeding, in order to eliminate a possible hereditary defect. In justice, it must be said, however, that Dr. Guyer did consult several animal breeders of wide experience and learned that they had never seen eye defects in rabbits. These men, however, were not ophthalmologists, and it is possible that occasional defects would have escaped their notice.

Our chief criticism of the work is that the ancestry of none of the three defective strains is known, all of the original females as well as some of the males having been received from other breeders without any definite knowledge of their pedigrees. Their earlier deductions with regard to the specific action on the lens was apparently unfounded, since the changes in the lens were secondary to faulty closure of the fetal ocular cleft. This phase of the subject has been discussed in detail by the senior author.[3]

A number of other workers have conducted experiments that attempt to reproduce the observations of Guyer and Smith, but up to the present time no one records similar results.

---

10. von Szily, A.: Die Ontogenese der iditypischen (erlbildlichen) Spaltbildungen des Auges, des Midrophthalmus und der Orbitalzysten: Ein Beitrag zum Problem der Vererbung und Erwerbung des Koloboms, Ztschr. f. Anat. u. Entwcklngsgesch. **74**:1, 1924.

## REVIEW OF OTHER EXPERIMENTS ATTEMPTING TO INDUCE EYE DEFECTS

Silfvast [11] knew of the work of Guyer and Smith and referred to it in his paper but made no specific attempt to repeat it. He injected pig lens and sheep serum immunized to pig lens into pregnant rabbits in an attempt to learn whether the immune bodies produced would pass through the placenta. He found that both active and passive immunization of the mother to lens substances causes the appearance of lens antibodies in the fetal blood. Incidentally ten litters were born to such immunized mothers, containing in all seventy-nine young, and these young all had normal eyes.

Finlay [12] injected lens sensitized fowl serum into pregnant rats and mice. His results are inconclusive, because he did not use rabbits, therefore not repeating the chief experiments of Guyer and Smith, and because most of the animals that received injections died or the young died. Too few young could be examined to allow for any definite conclusions regarding the production of eye defects. He observed no anomalies of the eyes.

Huxley and Carr-Saunders [13] injected lens antiserum from fowls into pregnant rabbits, treating twenty-nine animals and obtaining fifty-seven young, all normal. They also treated eleven pregnant rabbits with direct injection of pulped lens obtaining seventeen normal young. It will be noted that their number of experiments is large in proportion to the number of young obtained, indicating a high mortality of mothers after injection or a high ratio of abortion, both of which occurred. In the case in which the direct injections were made, several of the rabbits receiving injections of ox lens are recorded as dying in a state of shock. Careful analysis of the tables, however, shows that these animals could not have died in anaphylaxis, since the death followed the second or third injection, with intervals of only a few days between injections. It is our experience that anaphylaxis is extremely difficult to obtain in rabbits by the use of lens substance.

Poynter and Allen [14] passively immunized thirteen rabbits to rabbit lens, using fowls to produce the antibodies. From this series they obtained seventy-one normal young. They actively immunized six pregnant rabbits with rabbit lens, obtaining twenty-nine normal young.

11. Silfvast, J.: Ueber die Beziehungen des mütterlichen Organismus zum Embryo, Klin. Monatsbl. f. Augenh. **68**:815, 1922.

12. Finlay, G. F.: The Effect of Different Species Lens Antisera on Pregnant Mice and Rats and Their Progeny, Brit. J. Exper. Biol. **1**:201, 1923-1924.

13. Huxley and Carr-Saunders: Prenatal Effects of Lens Antibodies in Rabbits, Brit. J. Exper. Biol. **1**:238, 1924.

14. Poynter and Allen: Lens Antigen as a Factor in Congenital and Hereditary Eye Anomalies, Am. J. Ophth. **8**:184, 1925.

Two criticisms of their work may be made: First, they used dried lens substance instead of fresh lens, thus not following the technic of Guyer and Smith; second, they began injections on many of their rabbits near or after the time of closure of the fetal ocular cleft.

Ibsen and Bushnell [15] injected rabbit lens antiserum from fowls into ten pregnant rabbits and obtained fifty-nine young, of which one

TABLE 4.—*Summary of the Experiments of Various Workers Who Have Attempted to Induce Eye Defects in Rabbits by the Use of Lens Antigen and Uveal Pigment*

| Author | Method | Animals Treated | Total Young | Defective | Comment |
|---|---|---|---|---|---|
| Guyer and Smith.. | Fowl antiserum rabbit lens | 24 | 61 | 4 | 1 from female B; 3 from female 16A1 |
| | Rabbit lens; direct lens injection | 17 | 61 | 2 | Both defective, from same female 84 |
| Silfvast..... | Active and passive pig lens | 10 | 79 | 0 | Pig lens |
| Finlay..... | Fowl antiserum | .. | .. | 0 | Rats and mice; high mortality; only few young raised |
| Huxley and Carr-Saunders..... | Fowl antiserum | 29 | 57 | 0 | |
| | Direct lens injection | 11 | 17 | 0 | Several animals; injections of ox lens; died of shock |
| Poynter and Allen | Fowl antiserum | 13 | 71 | 0 | Used dried rabbit lens as antigen; many injections begun too late |
| | (Rabbit) direct lens injection | 6 | 29 | 0 | |
| Ibsen and Bushnell | Fowl antiserum (rabbit lens) | 10 | 59 | 1 | Slight cloudiness of lens |
| | Lens needling | .. | 186 | 1 | Bilateral small central opacity cornea |
| Davis and Smith... | Direct lens injection | 44 | 192 | 0 | |
| | Lens needling | 5 | 31 | 0 | |
| | Pigment injections | 12 | 60 | 1 | Hydrophthalmos: 3 animals of this series died in convulsions after injection of beef pigment |
| Totals (7)..... | | 181 | 903 | 9 | |

had a slight opacity of the lens. They also obtained one hundred and thirty normal offspring from pregnant rabbits whose lenses were needled and fifty-six from matings in which the eyes of both parents were needled. Among the latter, one had a slight bilateral central opacity of the cornea. We have also observed a case of corneal opacity, but have not recorded it, because it was clearly secondary to a conjunctivitis. The work of Ibsen and Bushnell seems to be thorough and lends considerable weight to our own conclusions. The foregoing experiments and their results are summarized in table 4.

15. Ibsen and Bushnell: Eye Defects in Rabbits, Anat. Rec. **46**:110, 1928.

SPONTANEOUS APPEARANCE OF COLOBOMA AND MICROPHTHALMUS IN THE RABBIT AS A HEREDITARY ANOMALY

The occurrence of coloboma in the rabbit has been known for many years. In 1880, Deutschmann [16] obtained a colobomatous offspring from a rabbit that had received injections of indifferent substances into the vitreous. The father of this animal had received an inplantation of tuberculous iris tissue into the anterior chamber.

Brown-Séquard [17] obtained high grade microphthalmus in several offspring of a guinea-pig the eye of which had been removed.

Samelsohn [18] carried iris tuberculosis through five generations of rabbits after transplantation of human tuberculous iris into the anterior chamber. Two animals of the fifth generation had bilateral microphthalmus. Their direct father (not related to the tuberculous line) had a unilateral anophthalmos.

Höltzke [19] described a rabbit eye with microphthaimus and coloboma, with cyst formation, which had appeared spontaneously.

Bach [20] described two microphthalmic fetal eyes which had presumably occurred spontaneously in breeding stock. He laid particular stress on the large, spherical lens, which we have found so common in colobomatous rabbit eyes and around which he attempts to build a theory regarding the origin of coloboma.

Von Hippel (1903) had a colobomatous male rabbit appear in his breeding stock which he afterward mated to normal females, obtaining twenty-three colobomatous fetal eyes out of one hundred and twenty-eight eyes examined. This paper contains a most interesting study of the nature of coloboma, the author concluding that the defect results from the persistence of mesodermal tissue in the fetal ocular cleft.

In 1907, von Hippel [21] again observed microphthalmus with coloboma in two families of rabbits that were born following the exposure of pregnant mothers to x-rays. In one of these cases, however, the

16. Deutschmann: Ueber Vererbung von erworbenen Augenaffectionen beim Kaninchen, Klin. Monatsbl. f. Augenh., 1880, p. 507.

17. Brown-Séquard: Faits nouveaux établient l'extrème fréquence de la transmission pour l'hérédité d'états organiques morbides, Compt. rend. Acad. d. sc. **94**: 697, 1880.

18. Samelsohn: Zur Genese der angeborene Missbildungen, speziell des Mikrophthalmus congenitus, Centralbl. f. d. med. Wissensch. **18**:305, 1880.

19. Höltzke, H.: Mikrophthalmus und Coloboma von eniem Kaninchen, Arch. f. Augenh. **12**:147, 1883.

20. Bach: Pathologisch-anatomische Studien über verschiedene Missbildungen des Auges, Arch. f. Ophth. **45**:1, 1898.

21. von Hippel, E.: Ueber experimentelle Erzeugung von angeborenen Star bei Kaninchen, nebst Bemerkungen über gleichzeitig beobachteten Mikrophthalmus und Lidkolobom, Arch. f. Ophth. **65**:326, 1907.

irradiation was given from the eighteenth to the twenty-third day of pregnancy, after the closure of the fetal ocular cleft, and, therefore, the defect cannot be attributed to the treatment. The second rabbit was exposed to the x-rays on the seventh, ninth and eleventh days of pregnancy, and several macerated colobomatous young were obtained in miscarriage.

Pagenstecher [22] recorded the appearance of unilateral coloboma with microphthalmus in a rabbit born from a mother that had been fed naphthalin. He attributed this anomaly to naphthalin poisoning of the retinal anlage, but it seems more probable that he was dealing with hereditary coloboma, especially in view of the fact that von Szily [23] has obtained five hundred and twenty-five young after naphthalin feeding, without a single case of coloboma.

Seefelder [24] published a short note regarding the material collected by Professor Hochstetter from 1904 to 1909, obtained by the mating of a colobomatous female rabbit and inbreeding the offspring. A large number of eyes was obtained showing coloboma, microphthalmus with cyst and lens opacities of various grades.

Koyanagi [25] obtained seventy-one colobomatous fetal rabbit eyes out of a series of two hundred and eighty-six eyes examined. These were obtained by breeding a colobomatous male rabbit to normal females and killing the females at definite times. His percentage of defects (24.8) is somewhat higher than those of von Hippel (18) and von Szily (21.9).

Von Szily [10] published an exhaustive article on the embryology of coloboma in rabbits. In this paper the work of Guyer and Smith is reviewed and criticized and a summary of the results of other investigators is given. He used three male rabbits in which coloboma had spontaneously occurred in a series of breeding experiments from 1909 to 1914, mating them to normal females. Inbreeding was not practiced. He obtained one hundred and three defective eyes out of four hundred and seventy studied, ranging from the eleventh day of fetal life to birth. In addition, von Szily stated that he has observed, in all, thirteen spontaneous colobomas in rabbits. Five of these occurred in one litter, of which two were bilateral; seven were in adult rabbits of different

22. Pagenstecher: Experimentelle Studien über die Entstehung der angeborenen Stare, Ber. über die 37 Versammlung der Ophth. Gesellsch., Heidelberg, 1911.

23. von Szily (footnote 10, p. 29).

24. Seefelder, R.: Ueber die Vererbung von Augenkolobmen, Ber. ü. d. Versamml. d. ophth. Gesellsch. **42**:210, 1920.

25. Koyanagi: Embryologische Untersuchungen über die Genese der Augenkolobome und der Mikrophthalmus mit Orbitalcyste, Arch. f. Ophth. **104**:1, 1921.

litters; and one unilateral coloboma occurred in a fetus. The parents of all of these were normal.

Ibsen and Bushnell,[15] in an abstract of their article, reported that three rabbits with noticeably defective eyes were born in their laboratory from untreated stock. One animal had a unilateral hydrophthalmos similar in appearance to the case we have reported. Dr. Ibsen sent us a photograph of this eye, although no histologic studies have yet been made. Another animal of this litter showed bilateral cataracts. They reported a third defective-eyed animal that showed a bilateral coloboma, with microphthalmus and opaque lens, which was born from normal parents. Two subsequent matings of the male, to his sister and to his mother, yielded one defective in each litter of seven and eleven, respectively.

We wish here to record the spontaneous appearance of coloboma in our own stock. We observed five colobomatous eyes out of twelve from a litter of six, the mother of which had normal eyes. She was pregnant when received in a shipment sent us by a breeder in Oshkosh, Wis. We had ordered this female mated before shipment so that we might more quickly obtain animals that could be used as a source of lens material for injections. This experience led us to secure other pedigreed stock for many of our subsequent experiments. This fortuitous occurrence furnished us at once with a known defective strain. This female was later mated to one of Dr. Guyer's males (123A6) that was outwardly normal but belonged to a defective strain, and produced two bilaterally colobomatous young in a litter of three. She was later mated to a normal male and produced eight normal young. Later attempts to mate this female failed, due to a vaginal inflammation.

A study of these recorded cases, especially those of Koyanagi and von Hippel, shows that coloboma is not a simple mendelian recessive, since it may appear in the first generation obtained from the mating of a a rabbit with defective eyes with a rabbit with normal eyes. Certainly it often occurs in a recessive form, however, as shown, since normal-eyed parents may produce colobomatous offspring. On the other hand, though both parents have defective eyes they may produce some normal offspring.

It appears, therefore, that coloboma is not caused by a single hereditary factor, but that at least two factors are involved. The facts, as far as known, seem to fit quite well the assumption that the condition is caused by a dominant factor for coloboma, but its action is normally prevented by a second factor—an inhibitor of coloboma. This mechanism adapts itself well to the explanation of the hereditary charts published by Guyer and Smith.

## CONCLUSIONS

The volume of negative results obtained by five well known investigators, combined with the negative observations of our own experiments, indicates that hereditary defects of the eye cannot be induced in rabbits by immunologic procedures directed toward the influencing of the young during fetal life. Hereditary anomalies of the eye such as were obtained in the experiments of Guyer and Smith are well known, and our own experience, together with that of a number of other observers, reveals that they not infrequently appear spontaneously in rabbits.

# OPTIC ATROPHY IN PAGET'S DISEASE*

RUDOLF AEBLI, M.D.
NEW YORK

The case presented is reported for three reasons: (1) because of the relative infrequency of seeing optic atrophy in Paget's disease, (2) because of the unusual ocular symptoms presented and (3) because the bony changes involved primarily the sphenoidal region and caused a partial obliteration of the optic foramina and to a lesser extent of the sphenoidal fissures.

The literature reveals few reports by ophthalmologists of complications of the optic nerve in Paget's disease. Sir James Paget [1] first described this disease before the Medico Chirurgical Society in 1876, and gave a complete picture which has not been improved on. Lawford R. Knaggs, in February, 1925, in the Hunterian Lecture before the Royal College of Surgeons in England, said that Paget's disease is "A very chronic disease of an inflammatory nature occurring in middle age or elderly people in which the normal osseous structure of the skeleton is removed, and fresh bone formation on a different plan and in a larger mould takes its place. It is the bone changes that constitute the important and characteristic features of osteitis deformans." Goldstein,[2] in his comprehensive review, stated that there are about four hundred recorded cases of Paget's disease in the literature. He reported twenty cases in all of which there was a moderate degree of deafness, marked arteriosclerosis, negative Wassermann reactions and normal blood sugar and uric acid.

In the ophthalmic literature only a few cases of involvement of the optic nerve have been recorded. Wyllie [3] reported two cases of optic atrophy in osteitis deformans. In both cases there was a partial bilateral optic atrophy of slow progression with a gradual reduction of vision to 10/200. No x-ray films of the foramina were taken, but the author suggests narrowing of the optic canals as the cause. Pincherle [4] reported a case of osteitis deformans in which there was paralysis of

* Submitted for publication, June 18, 1930.

* Read at a meeting of the New York Academy of Medicine, Section of Ophthalmology, Jan. 20, 1930.

1. Paget, James: Osteitis Deformans, Med. Chir. **60**:37 (Nov.) 1876.

2. Goldstein, H. J.: Paget's Disease of the Bones, M. Times **50**:279 (Nov.) 1922; 321 (Dec.) 1922; **52**:56 (March) 1924; **54**:194 (Aug.) 1926.

3. Wyllie, W. G.: Proc. Roy. Soc. Med. (Sec. Ophth.) **17**:45, 1923-1924.

4. Pincherle, P.: Zentralbl. f. Ophth. **18**:870, 1927.

the left external rectus with occlusion of the left sphenoidal fissure and narrowing of the optic canal with atrophy of the nerve.

In the case that I shall report there was a complete right optic atrophy with vision reduced to a small island of perception of hand movement in the upper part of the field and a partial left optic atrophy with vision reduced to 20/200. The roentgenograms made by Dr. Harry Goalwin [5] show the entire sphenoidal bone to be markedly

Fig. 1.—Paget's disease, showing moderate enlargement of vault, fusion of tables, multiple areas of sclerosis alternating with areas of rarefaction. The entire sphenoidal bone is sclerosed and thickened.

sclerosed and thickened. Both optic canals were triangular and constricted, the right more so than the left. The diameters of the optic canals are: right, 4.3 by 2.6 mm.; left, 4.3 by 3.1 mm. The former is below the minimum compatible with normal vision; the latter is the minimum normal.

5. Goalwin, Harry: Precise Roentgenography and Measurement of the Optic Canal, Am. J. Roentgenol. **13**:480, 1925.

REPORT OF CASE

*History.*—A man, aged 68, first consulted me in July, 1928. He said that the night before when returning home from work he noticed that the right eyeball was red, that the lids were swollen, and that that eyeball was more prominent than the left. He had experienced some diplopia during the day, but as he had no pain

Fig. 2.—Roentgenogram, showing normal optic canals.

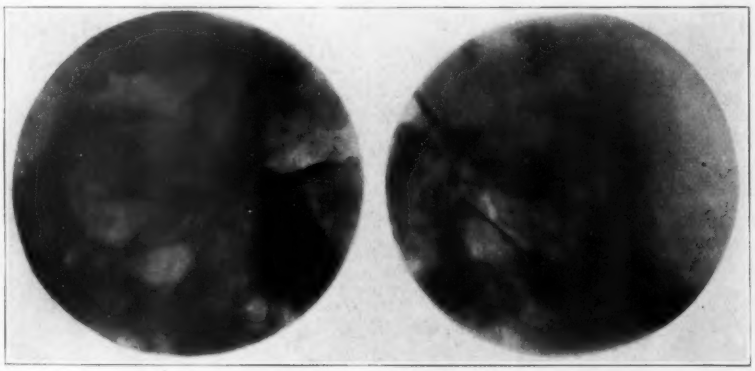


Fig. 3.—Roentgenogram, showing right optic canal triangular and constricted below the minimum compatible with vision; left minimum normal.

and could see with the eye he paid no particular attention to it. The following morning when he awoke the swelling and redness and the protrusion of the right eye had increased, and then he observed that he had no sight in the right eye. He had no pain in the eye and no physical discomfort. Prior to the onset of the present condition he had worked continuously as an elevator operator for twenty years. Eight years previously he noticed that his hearing was impaired on

the left side, and at that time he sought advice in one of our clinics. The cause of the defective hearing was not discovered. He stated that aside from his deafness he had always been in good health. He had observed that during the past few years he had grown considerably shorter, and that he was now 6 inches (15.24 cm.) smaller. He also stated that it was necessary to have his derby hat stretched on two occasions, as it was gradually becoming too small for him.

*Examination.*—Physical examination showed a marked kyphosis with a large head and coarse features. The heart and lungs were normal; the Wassermann reactions of the blood and spinal fluid were negative, and the chemical analysis of the blood gave negative results. Roentgen examination showed the bony changes characteristic of Paget's disease.

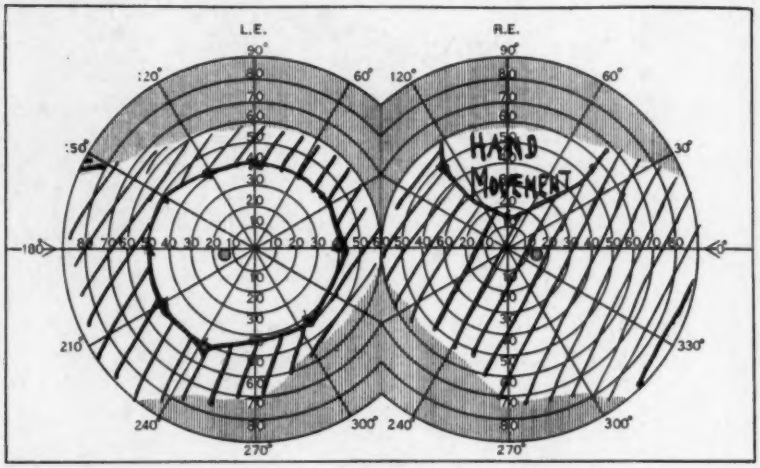



Fig. 4.—Visual fields, taken on Jan. 2, 1930.

There was a marked unilateral exophthalmos of the right eye with edema of the upper and lower lids and of the palpebral and bulbar conjunctiva. There was a complete loss of motility. Vision was reduced to the perception of hand movements in the upper field. The tension was normal, there was no pain on palpation, and with gentle pressure the eyeball could be pushed back into the socket. Examination of the fundus showed marked engorgement of the veins, contracted arteries and an atrophic nerve head. The left papilla was pale; vision was reduced to 20/200 with a concentric contraction of the field.

*Progress.*—In the course of two weeks the edema gradually subsided, the eyeball receded into the socket, and the motility returned. There was complete loss of vision, except for a small island of perception of hand movement. The patient has now been followed up for eighteen months. The condition in the right eye has remained about the same. In the left eye the vision is gradually growing worse, and the field is slowly contracting. Except for his impaired hearing and poor vision, the patient's general health has remained good.

# THE SIGNIFICANCE OF SOME CHANGES IN THE VESSELS OF THE FUNDUS*

ARTHUR J. BEDELL, M.D.
ALBANY, N. Y.

So much is heard about arteriosclerosis and high blood pressure that this study was undertaken to see whether it was possible to draw definite conclusions concerning these symptoms from an inspection of the eyegrounds and a critical analysis of a large number of photographs.

There are all grades of physiologic vascularization of the fundus. The course of the vessels presents unlimited variations not only in the number, position and size of the major subdivisions, but also in their course and contour. The vessels of some fundi are almost straight, and in others twisted convolutions like a medusan head cover the field. An essential characteristic of a normal vessel is that its caliber is unchanged at vein and artery crossing.

As most physicians know and as all ophthalmologists appreciate, the fundus is the scenic background on which is thrown life in its various phases of vascular change. By means of the photographing ophthalmoscope, I have observed many things in a new light; I have studied the pictures in a receptive mood and in an atmosphere of complete isolation. The marvels of stereoscopic photography of the fundus have so impressed me that I have often seen the commonplace transformed into the unusual, and the interpretation of a picture sometimes read like an inspired revelation.

Two particular vascular catastrophes present completely dissimilar pictures: thrombosis of the central vein and sudden closure of the central artery. The latter has for years been called embolism, but recently a questioning note has been sounded, so that one must view the subject primarily as the closure of the artery and try to distinguish photographically between that which seems to be a frank embolus and the other type which probably is of thrombotic origin secondary to alterations in the wall of the blood vessels. Although this clinical differentiation has not been proved pathologically, it seems to me to have something in it of sufficient value to warrant its presentation.

---

* Submitted for publication, Oct. 22, 1930.

* Read before the Section on Ophthalmology at the Eightieth Annual Session of the American Medical Association, Portland, Ore., July 11, 1929.

### TYPICAL CHANGES IN ARTERIAL CLOSURE

In the rapid shutting off of the arterial circulation there is a blanching of the retina supplied by the occluded vessel. This pallor of the fundus is followed by a period of haziness of the retina, called edema by some and a coagulation necrosis by others. If the occlusion is of an isolated vessel or if the center of the retina is supplied by a cilioretinal vessel, the margin of the part affected is of a thick milky white color. This is the last portion to resume its normal color after the collateral circulation is established. Because of the proximity of the normal and the pathologic, this is the best region in which to study the onset and subsidence of the process. The occluded artery is always smaller and whiter than normal. When the circulation is restored or carried by collaterals, an interrupted blood stream is observed. This may become a continuous current or be entirely abolished. The veins are always decreased in caliber at the onset and reach their normal size only when the retinal circulation is adjusted to the patent vessels.

In total closure of the central artery the foveal area is by contrast a dark red. As the blood returns, that is, in a few days, the reddened area is reduced in size; but as the venous radicles extend, and this is the stage when the so-called edema is thickest, the dark macular zone increases in size with an elevated pallid surrounding wall.

Later the macula flattens and small yellow spots appear in the retina in front of or about the fovea. Coincidentally, the optic nerve assumes the startling white color of atrophy. In the terminal stage of rapid closure, the arteries are small, white threads, the veins are narrow, the disk is white, and scattered over the macula are many small, yellow, discrete dots. These dots are exactly the same in structure as those seen after the formation of a hole in the macula and are therefore of considerable interest.

In the subtotal forms, the contracted artery and the change in texture of the retina are constant permanent observations.

The accompanying illustrations show three types of arterial occlusion: total, in which the classic clinical signs are present; partial, in which one vessel is closed, and a third, in which an auxiliary system preserves the central field. The group that really suggests the thrombosis theory is one in which there is evidence of rapid occlusion with, however, superficial retinal hemorrhages; as hemorrhages accompany venous thrombosis, it may be possible that this gives a clue to the process transpiring within the artery.

In the study of vessels, it is also interesting to note that after the blockade the arteries sometimes become smaller and shrink; in other cases streaks of periarteritis develop, and in still others there are the white plaques suggestive of nutritional changes in the vessel wall itself.

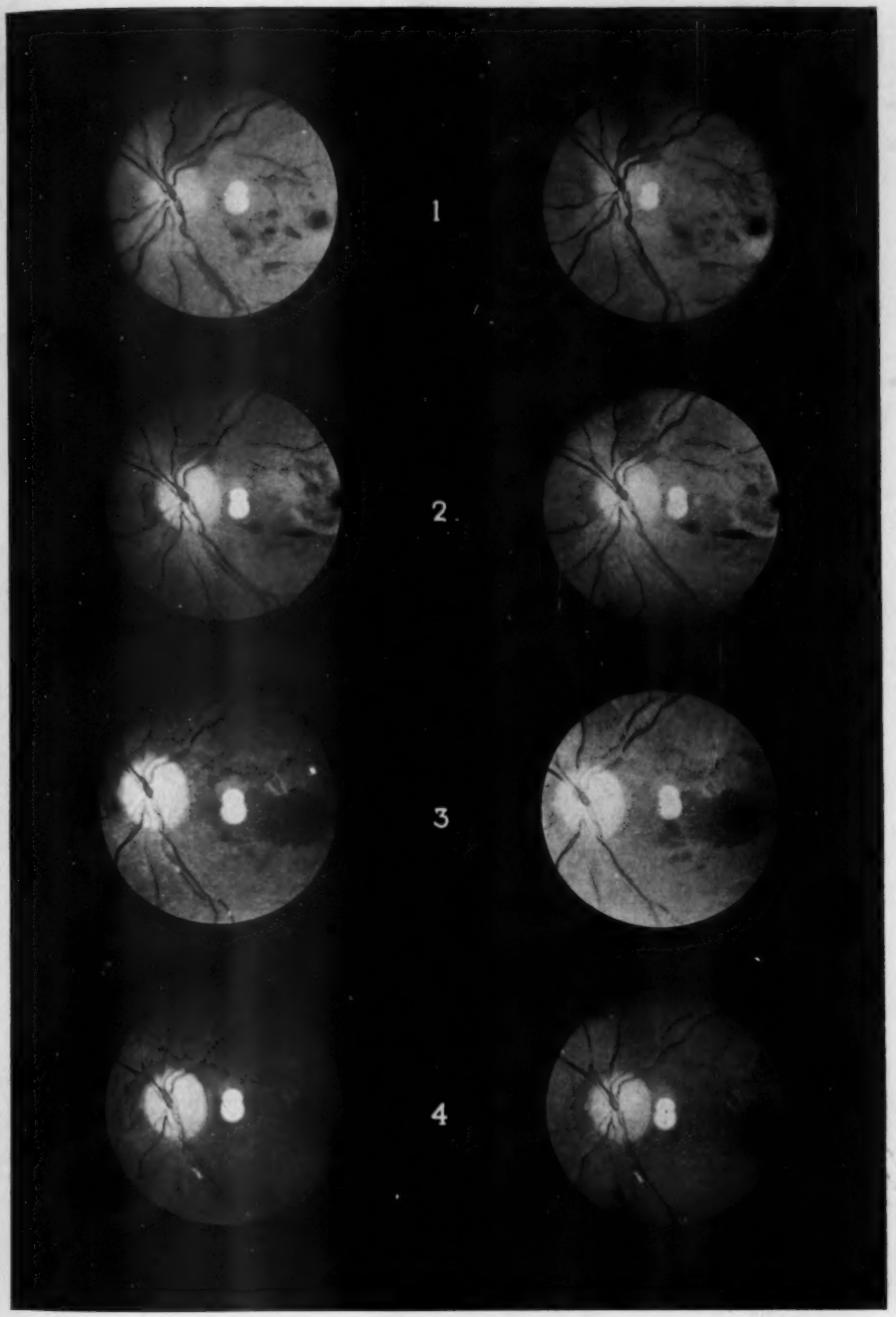



Fig. 1.—Sudden closure of the central retinal artery (thrombosis?).

The following case illustrates some of these points:

CASE 1.—A woman, aged 67, had had high blood pressure for ten years with considerable thickening of the peripheral vessels and chronic arthritis of both hands. The blood pressure ranged from 220 systolic and 120 diastolic to 230 systolic and 140 diastolic. The urine showed a specific gravity of 1.014 with a slight trace of albumin. The Wassermann reaction was negative.

Ten days before the first view was taken (fig. 1, *1*), the patient noticed that the left eye was lacrimose, and the next morning she found that it was blind. The illustration is of unusual interest because it shows not only the marked retinal edema so often found in rapid closure of the central artery but the various levels of hemorrhage. The blood was spread in many layers between the disk and the macula. The macula itself was covered by a dark red oval clot. The vessels were smaller than normal; their caliber was unequal; in places the artery was much reduced in size and in other regions it was almost its former width.

Ten days later (fig. 1, *2*) the edema had lessened, but it remained greatest between the macula and the disk. All the vessels were reduced in size and number, the arteries showing the greater change. The macular region was swollen and gray, with blood flecks and large clots on its surface. A pale, circular outline with fine vessels on its upper edge had a diamond-shaped clot as a part of its lower margin.

Forty-seven days after the onset (fig. 1, *3*), the disk was white, and the number and size of the vessels showed the steadily progressive shrinking streaks of periarteritis and several white plaques appeared in the artery wall. The macular edema had subsided, leaving a round, granular, red area of blood above which the white arterial twigs were visible.

On the sixty-eighth day (fig. 1, *4*), the degeneration had progressed; there were fewer vessels, all of reduced caliber, and an increased number of larger white plaques. The granular blood was being absorbed and the perimacular white artery lines were increasing.

### THROMBOSIS OF THE VEIN

The other calamity is thrombosis of the vein. The appearance of the fundus depends on the size and position of the obstructed vessel. In small subdivisions the blood seems to seep through the wall; most often yellowish-white flocculent exudate masses are associated with it, so that the blood either lies on, or perhaps more often becomes mixed with, the exudate. In this series of cases the site of predilection was the superior temporal branch, less often the inferior temporal and rarely the superior nasal and inferior nasal. The typical clinical history of such cases is well known. In the fortunate patient the blood disappears, function is restored and no gross trace of the incident remains, as figure 2 demonstrates. It is, however, possible to have the thin wall sufficiently altered to enable one to draw the conclusion that it was formerly surrounded by a massive hemorrhage.

Total thrombosis of the central vein is characterized by the spattering of blood through the retina, the edema of the retina, the partially veiled, bright-walled arteries and the preservation of the central excavation.

This condition is seen too often to warrant much detailed description, and yet, because of the gloomy prognosis often given and especially the described tendency to secondary glaucoma, attention is drawn here to some of the discriminating features of the various types as I have seen them photographically, in the belief that the illustrations will help to separate primary thrombosis of the central vein from hemorrhagic glaucoma. Clinically there was such great variance in the appearance of the fundus in this disease that I studied the records in the hope that an accurate prognosis might be given. But my conclusions do not warrant any pronouncements, for I found in partial thrombosis that the blood pressure was low or normal in one half of the cases and high in the other 50 per cent. In the cases of total occlusion, the blood pressure was definitely elevated in 66 per cent, and low or normal in the remaining 34 per cent. The ultimate outcome of thrombosis depends on the location of the block, the age of the patient, the condition of the general blood vessel system and the accompanying local disease.

The unilaterality of thrombotic processes is well shown in figure 2.

CASE 2.—A woman, aged 68, noticed a blur on the inner side of her eye thirteen days before examination. The systolic pressure was 108. Examination of the left eye showed a vision of 20/50.

The irregular light reflex from the arteries seen in the first view (fig 2, *1*) was due to caliber change and the angle at which the vessel was observed. The large excavation of the disk afforded an excellent view of the deep vessels.

In the right eye, vision was 10/200. The clear disk had a large, deep, central excavation (fig. 2, *2*). The veins were broad and fuller than normal. In the lower half of the field was a thick mass of blood through which the artery twisted its way, leaving a comparatively clear channel. In the lowest field shown, the dark area is a preretinal hemorrhage.

In the third view (fig. 2, *3*), the outer, temporal region shows the blood as a large, flat-topped hemorrhage anterior to the retina. The artery can be traced to the upper edge of the extravasation and is seen again at the outer side. A few white arterial branches are also visible.

When the patient's head was tilted the blood between retina and vitreous sought a new level, but it was so fluid that before the camera could be adjusted and the photograph taken, much of it returned to the primary position. The elongation of the dark area (fig. 2, *4*) demonstrates, however, its fluidity and preretinal position.

This is an excellent example of thrombosis with moderate arteriosclerosis and hypotension.

In the Scientific Exhibit, pictures are shown of a patient who had glaucoma on one side and on the other a total thrombosis of the central retinal vein, from which he recovered with vision of 20/20. It will also be observed that the tendency in all these cases seems to be toward recovery. The blood is absorbed and, if new hemorrhages are prevented, sight is restored.

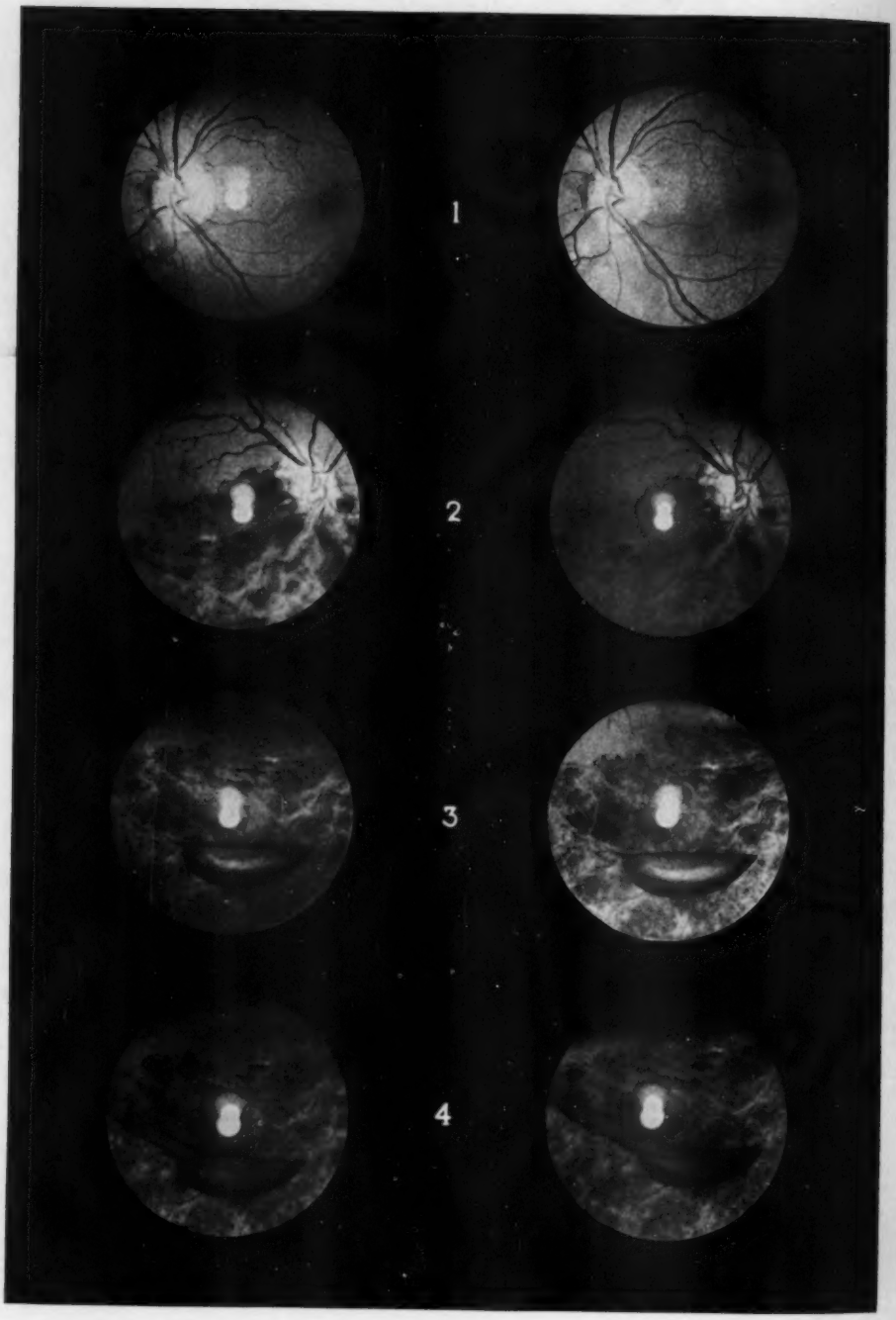



Fig. 2.—Thrombosis of a retinal vein: preretinal blood.

Before this subject is closed, particular attention is called to the behavior of the individual vein and artery, as may be noted by inspection of the separate illustrations, for the cases group themselves as follows: (1) The veins are large, full and tortuous with normal arteries; (2) exudates are spread about the vessels and the hemorrhages, and (3) although the blood is well scattered over the fundus, the veins are not markedly distended and the exudate is practically absent.

### RETINITIS PIGMENTOSA

It may be possible to make a differential diagnosis between retinitis pigmentosa and retinochoroiditis by the study of the retinal arteries. This can be more easily appreciated by the examination of figures 3 and 4.

CASE 3.—A woman, aged 67, had a vision of 20/20 in each eye, with a correcting glass. She had always suffered from an extreme nyctalopia.

In the right eye (fig. 3, *1*), vision was 20/70 + 1.00 = + 50 × 90 degrees, 20/20. The nasal side of the eye showed a pale gray disk on a silvery gray retinal background. All the vessels were small; some of the arteries were mere threads. The pigment was found along the vessels and separated from them but always anterior to the choroid.

The central vein of the same eye (fig. 3, *2*) gave little evidence of the great peripheral pigmentation. The pale region seen about the disk and below it was visible choroid.

In the left eye (fig. 3, *3*), vision was 20/40 + 75 = + 50 × 90 degrees 20/20. The posterior pole on the left eye presented tortuous corkscrew vessels, and a zone of pigment absorption about the disk with little pigmentation. The disk itself was flat and dull gray, but the surrounding choroid was almost glistening in its light reflections. The pigment was in front of the choroid. All the arteries were smaller in proportion than the veins.

In the nasal side of the same eye (fig. 3, *4*) were very small, thick-walled arteries, some of them mere grayish-white lines. The veins remained of fair size. The pigment was collected in dense black masses of round and linear outline anterior to the choroid.

### SYPHILITIC RETINOCHOROIDITIS

The distinguishing characteristics of the blood vessels in syphilitic retinochoroiditis are shown in the two eyes illustrated in figure 4.

CASE 4.—A man, aged 41, with a vision of 20/50, had had a Wassermann reaction of + + + + for five years.

The disk was pale but clearly outlined (fig. 4, *1*). The fields in the upper half of the fundus were narrowed, and the arteries were especially contracted on the disk and for a short distance beyond. When they again widened out, the lower vein was almost of normal caliber. The accompanying artery was straight. The upper part of the fundus was depigmented silvery gray with small flecks of migrated pigment. There was an ovoid patch of choroiditis above the macula.

In the nasal half of the same fundus (fig. 4, *2*) the arteries were attenuated and the veins small. Many collections of black pigment assumed a ring form,

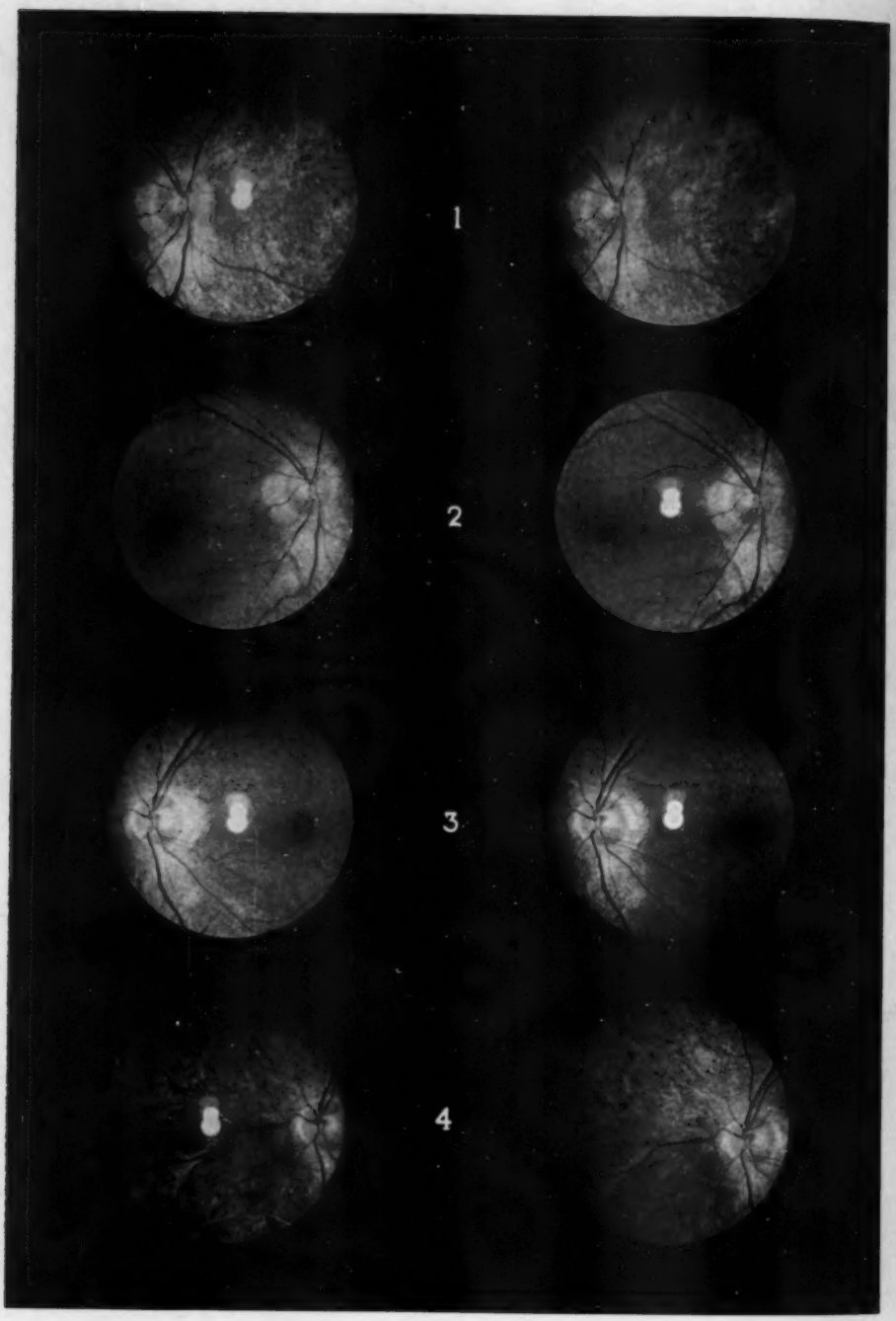



Fig. 3.—Retinitis pigmentosa.

while others were without any characteristic outline. The background was mottled, in places very pale and in others almost of normal color.

CASE 5.—In a man, aged 35, nyctalopia developed eight years before examination. The Wassermann reaction was ++++. Vision was 7/200 — 2.00 = 20/30. There were several posterior cortical globules (fig. 4, *3* and *4*). The disk was almost of the same color as the background, but a narrow scleral ring served as a demarcating border. The central excavation was filled in with a gray, cloudy exudate. The arteries and veins were both reduced in size. The background was pale with considerable pigmentation, usually in the form of small, black dots. In the fourth picture the temporal region was shown; the macula was well outlined; the pigment was collected in indefinite circles, and the whole fundus was bespeckled. The feature to which particular attention is drawn is the size of the veins. This is a valuable point in separating this disease from retinitis pigmentosa.

When retinitis pigmentosa has existed for many years the dirty gray disk is faintly differentiated from the gray light-absorbing fundus. This makes a dull picture. The arteries may appear only as dark threads on the disk or, if the perivascular pigmentation is excessive, as black lines. The extreme case shows the white narrow arteries and dark vein walls. This contrasting difference of color seems to remain as long as there is any visible circulation of retinal blood. The arteries may become so attenuated as to be scarcely perceptible and are frequently the site of a complete black invagination. This tube may be interrupted; it may extend more centrally than the rest of the pigmentation, and not uncommonly a depigmented zone is contiguous with it. The tubal form of pigmentation may be extremely thin and photographically demonstrable only at vessel crossings; at other times, however, the pigment gives the impression of a real widening of the vessel. Occasionally a narrow black cylinder has a pale border, or it may even consist of small pigmented nodules. The most contracted vessels are usually in the field of the greatest migration of pigment. Gray lines with light reflexes below them are associated with the accumulation of pigment in the more distal portions of the same vessel.

### CLINICAL VARIATIONS IN VISIBILITY OF CHOROIDAL CIRCULATION

The choroidal circulation, which is visible only in partial or complete albinism, in myopia, in the sclerosis of disease and of injury and also in detachment of the retina, may now be briefly considered. The bizarre distribution of the vessels and the location of the venae vorticosae and collecting systems all tend to clinical variations which at times are difficult to interpret. The examination of a few so-called normal eyes is therefore necessary before one can see the sclerosis which is described by all pathologists and probably is invariably present, but is infrequently seen and is less often pictured.

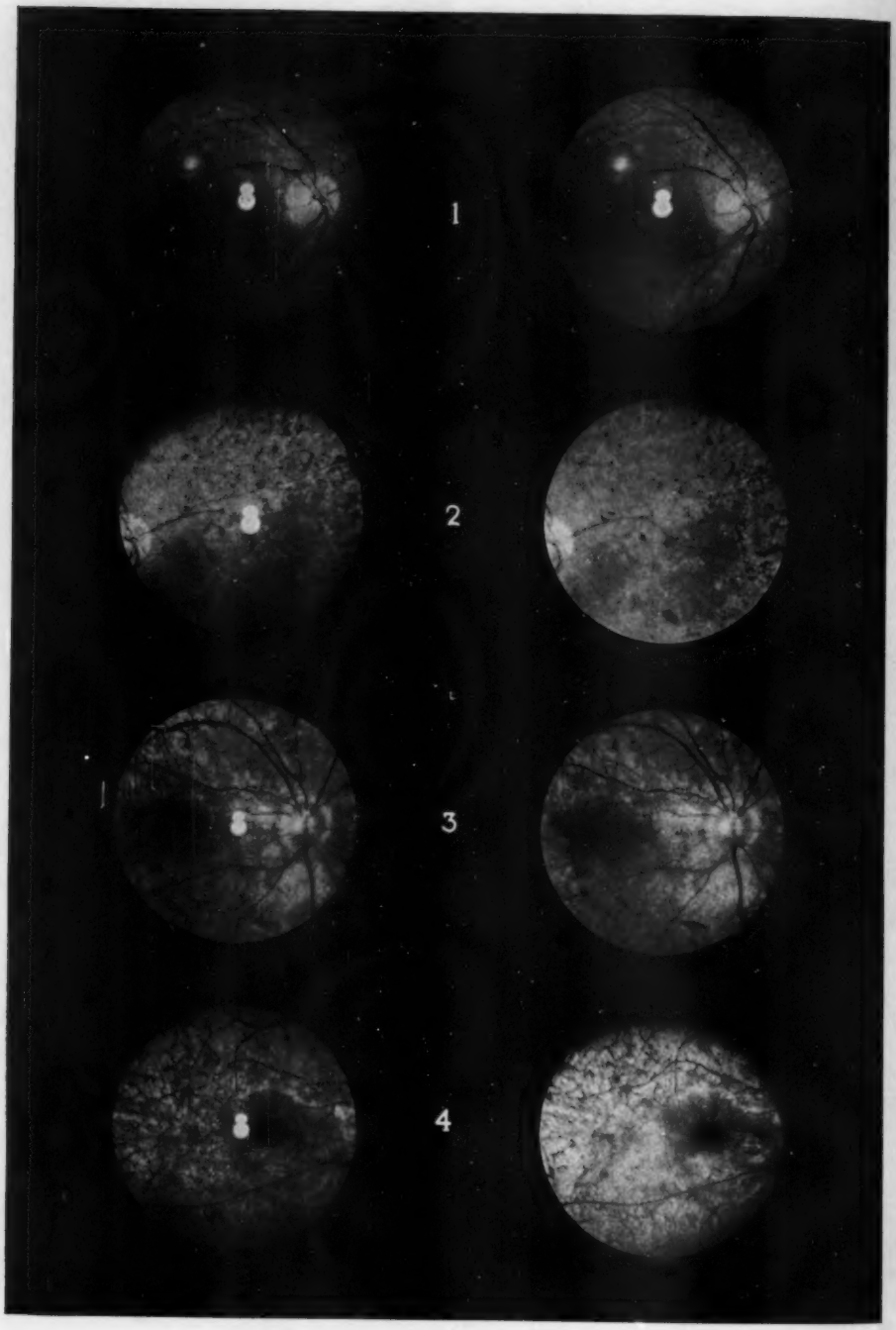



Fig. 4.—Syphilitic retinochoroiditis.

In the Scientific Exhibit many illustrations were presented. The one illustration presented here (fig. 5) so clearly emphasizes certain features of the problem that the clinical histories are appended:

CASE 6.—A man, aged 53, who showed a marked quadrant defect in the field of vision, had had a Wassermann reaction of ++++ for several years. Vision was $5/200 - 5.00 = -75 \times 105$ degrees 20/20.

The lower edge of the disk (fig. 5, *1*) was rounded and elevated; a slight change was found in the lower arteries. The upper branches showed white streaks of periarteritis and some occlusion with obliterated terminals. There was irregular pigment absorption with three large, thick, black plaques. The change was more marked in the periphery of this upper sector, where several very bright dots were seen in the retina. The upper nasal border of the macula was an elevated grayish-yellow ridge of exudate.

CASE 7.—A woman had been under my personal observation for twenty years. During that time the condition had remained unchanged. It was of undetermined origin. The blood pressure was 146 systolic and 100 diastolic. Vision was $5/200 - 3.00 = -50 \times 90$ degrees 5/200.

A sharply outlined area of pigment absorption (fig. 5, *2*), whitish near its upper boundary, allowed the choroidal vessels on the scleral base to be seen. All the retinal vessels were smaller than normal. The disk was pale and the macula granular.

CASE 8.—A man, aged 64, with posterior cortical cataract, showed the same condition. The disease of the fundus had been present for at least thirty years. Vision was 1/200.

About two thirds of the region surrounding the pale disk was depigmented (fig. 5, *3*). The retinal vessels were small and only a few minor branches remained. The white, sclerosed choroidal vessels were visible, although partially covered by pigment in places.

CASE 9.—A man, aged 59, had a high degree of myopia. Vision was limited to perception of light.

This case furnished the best illustration (fig. 5, *4*) of sclerosis of choroidal vessels that I have photographed. A dark lower crescent was all that showed as retina with the exception of the attenuated retinal vessels. Patches of pigment concealed some of the white, sclerosed vessels, but where there was no overlying pigment the twisted mass and the different layers were well shown. Careful fixation demonstrated the various levels.

### CHANGES CHARACTERISTIC OF NEPHRITIS

In nephritis the vessel walls may show slight changes amounting to nothing more than an increased visibility and vein compression. Of course, it must be remembered that in chronic nephritis the arteriosclerotic factors may be the most prominent ones.

The changes in the fundus in this form of nephritis are so well shown in figure 6 that little comment is necessary, yet if one is to believe that the radiating lines of exudate about the macula are to be found in several disease conditions one's time is well spent in differentiating the types of macular star. Three distinct groups are portrayed. The first

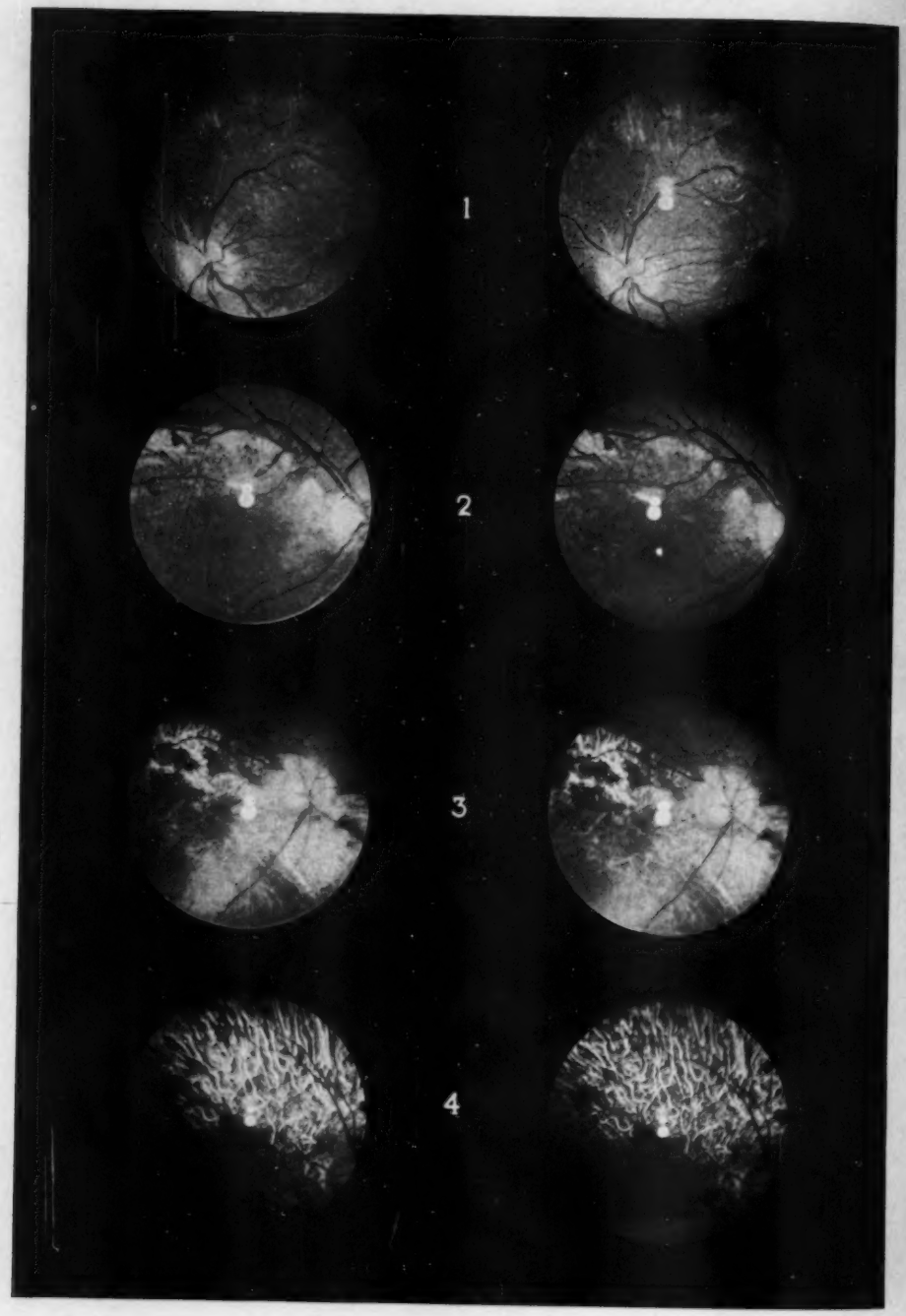



Fig. 5.—Choroidal sclerosis.

view in figure 6 shows broad, bright yellow streaks; the second, finely granular yellowish lines, and the third and fourth, almost white massive radiations. All, however, are renal.

CASE 10.—A colored man, aged 39, who was suffering from acute nephritis with a systolic pressure of 190, complained only of blurred vision.

In the eye not shown there were large, dirty-white snow banks of exudate. In the left eye (fig. 6, *1*), the vision was 20/40. The pallid disk, the moderate increase in the visibility of the artery wall and in vein compression, combined with the superficial hemorrhages and exudate and the deep radiating retinal exudate, make a classic picture of renal retinitis.

CASE 11.—A man, aged 34, with chronic nephritis which proved rapidly fatal, death occurring within five months of his original complaint, showed a contrasting picture. His systolic blood pressure was 200. Vision was 4/200. An eccentric view (fig. 6, *2*) presented the yellowish-white reflecting deep exudate arranged as a star. There was vessel compression and edema of the retina. Capillary varices were seen about the macula overlying the exudate.

CASE 12.—In a man, aged 31, a fog came before the eyes, five months before examination. He showed an excessive amount of albumin in the urine; no concentration by the Mosenthal test; nonprotein nitrogen, 50; creatinine, 2.72; uric acid, 4.60, and a systolic pressure of 185.

In the eye shown in figure 6, view *3*, vision was 20/50. General edema of the entire retina obscured the arteries and veins and veiled the disk. The vessels were reduced in size. Several superficial flecks of hemorrhage were observed. Masses of deep white exudate radiated from the macula about which they had collected.

In the other eye (fig. 6, *4*), vision was 20/40. A great generalized edema obscured all the details of the fundus. The vessels could be outlined and flecks of superficial blood distinguished. A deep whitish exudate was massed about the macula.

## DIFFERENTIATION BETWEEN ARTERIOSCLEROSIS AND HYPERTENSION

I have intentionally taken some striking examples of changes in the retinal blood vessels, so that in their presentation attention might be drawn to the individual characteristics of each group and the way prepared for a short clinical discussion; in fact, a preliminary communication on the subject of distinguishing the actual alterations in the blood vessel walls, which up to this time have been more or less confused whenever hypertension and arteriosclerosis have been investigated.

It should therefore be understood that arteriosclerosis and hypertension are not synonymous terms. Arteriosclerosis is a condition produced by several gross causes. Further, arteriosclerosis is as often found in normal or subnormal pressure states as in hyperpiesia. By using two instruments—the ophthalmoscope and the sphygmotonometer—it is possible to make a differential diagnosis between primary arteriosclerosis and hypertension.

It is probable that no ophthalmologist would concede that he did not understand all of the fundal manifestations of this protean disease,

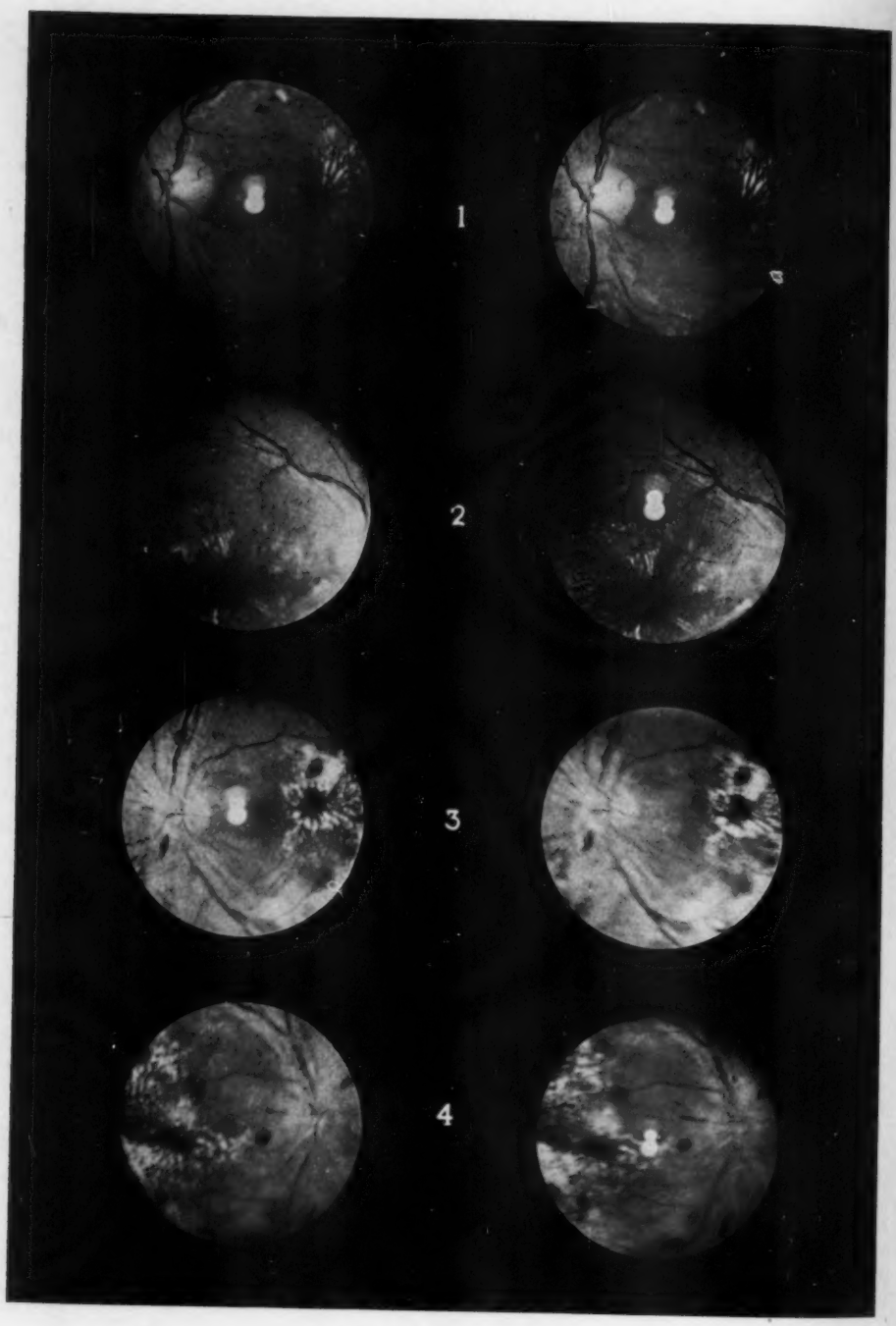



Fig. 6.—Renal retinitis.

but nevertheless it is essential for the clarity of the demonstration that the clinical signs of sclerosis of the retinal vessels be reviewed. Before this is done, attention is called to the appearance of the retinal vessels when viewed at different angles and under different light conditions. To master abnormalities completely one must have a firm foundation of the physiologic deviations. If one is to give a correct opinion he must remember the source and strength of the light used, the size of the pupil, the clearness of media and the age of the patient. It is because the work is performed under a known, constant light that photographs are making an appeal to those interested in scientific records.

### CHANGES IN ARTERIOSCLEROSIS

The primary change in arteriosclerosis is a pushing aside of the vein where the stiffened artery parallels it. Usually at this time there is what seems to be an increased reflex from the arterial blood, which shows as an interrupted bright streak, called the copper wire reflex. When the process is more advanced the artery causes either a faint denting or a trifling elevation of the vein, depending on whether the artery is behind or in front of it. The most accessible region for an early recognition of this symptom is the course of the temporal vessels, where the first artery crossing is usually sufficiently distinctive; occasionally, however, the vessels must be traced for some distance into the periphery before they cross.

Later the progressive hardening of the arterial wall produces a deep depression in the crossed vein, or uplifts it. During all of this time, in a large number of cases, there is no great alteration in the macular circulation.

When the venous compression is marked, there is an increase in the tortuosity of the vessels, a dilatation of the distal portion of the vein with ampuliform aneurysms, and frequently minute dilatations in the vessels suggesting a string of beads. The more advanced the process, the more opaque the arterial wall becomes, so that when the artery crosses the vein there is a wide streak in which the underlying vein cannot be seen. This is rather an important symptom and one that not only is shown in the pictures but is of striking beauty in the living fundus.

If the sclerosis continues, there will be one of two distinctive changes: an increased tortuosity of the arteries or a marked contraction of them. When there is a general atrophy of the vessel itself, it is not often possible to see the plaques develop in the walls. The whole vessel loses its transparency and in the advanced stages presents as an almost uniformly wide gray or white line. In the tortuous arteries, one finds a great increase in the burnished appearance of the surface, and later the development of telangiectases. Figure 1, illustrating the probable thrombosis of the central artery, gives a good idea of the

plaques as they develop in the artery wall and the white lines as they are produced about the macula.

Thin, white lines may be found on the sides of some arteries. These are, of course, perivascular lymph sheaths of increased density and, therefore, more visible. The lines may become fused by encircling the vessel, and the result is narrow, broad or disproportioned white tubes. When they are strongly light-reflecting, almost brilliant white, they are called pipestem arteries or atheromatous vessels.

### TYPE OF RETINAL ARTERIOSCLEROSIS

Because of its rather rare occurrence, practically complete sclerosis of the retinal arteries is here presented under three divisions: syphilitic, nonsyphilitic and the result of pernicious anemia.

CASE 13.—A man, aged 38, had been under treatment in many hospitals in the East. Vision was 1/200. For three years, vision had been steadily failing. The Wassermann reaction was ++++.

The extensive sclerosis of all the retinal arteries (fig. 7, *1*), with a brilliant white streak temporally, was intensified by the streaks of blood along some of the vessels. A large, superficial granular blood mass lay over the macula; about this the fine, white arteries were found. The veins of the fundus were small. The disk was white with a clearly defined border. Photography was difficult because of vitreous and lenticular opacities.

CASE 14.—A woman, aged 22, began to have sore eyes, four years before examination, following a nervous collapse which was caused by the sudden death of a near relative. A searching investigation failed to disclose the underlying cause. Vision was 20/100. The Wassermann reaction was negative. During the past four years the patient has had several attacks of red eyes.

In one eye (fig. 7, *2*), the extreme sclerosis of all the retinal arteries, the periphlebitic lines, the white nodes of anastomosis about the obliterated arteries, the white atrophic disk and the streaks of blood made a striking picture of almost complete obliteration of the vessels. . . granular hemorrhage was present in the macular region.

In the other eye (fig. 7, *3*), vision was 20/70. The blood pressure was 100. There was practically complete sclerosis of all the vessels. The white nerve showed the atrophic cup. On the grayish-white fundus all details of the retina were lost and the macula was covered by an irregularly outlined blood mass.

CASE 15.—A man, aged 54, presented, on November 23, a blood pressure of 110 systolic and 70 diastolic, with the following blood picture: The red blood cell count was 2,408,000; hemoglobin, 50 per cent; white blood cell count, 7,000; polymorphonuclears, 65 per cent; lymphocytes, 20 per cent, and endotheliocytes, 15 per cent.

On November 25, the hemoglobin was 65 per cent; the red blood cells numbered 1,120,000, and the white blood cells, 6,600. Liver therapy was started.

On November 29, the hemoglobin was 65 per cent; the red blood cells numbered 1,924,000, and the white cells, 4,600.

On December 22, the hemoglobin was 70 per cent; the red blood cells numbered 4,112,000, and the white cells, 6,200.

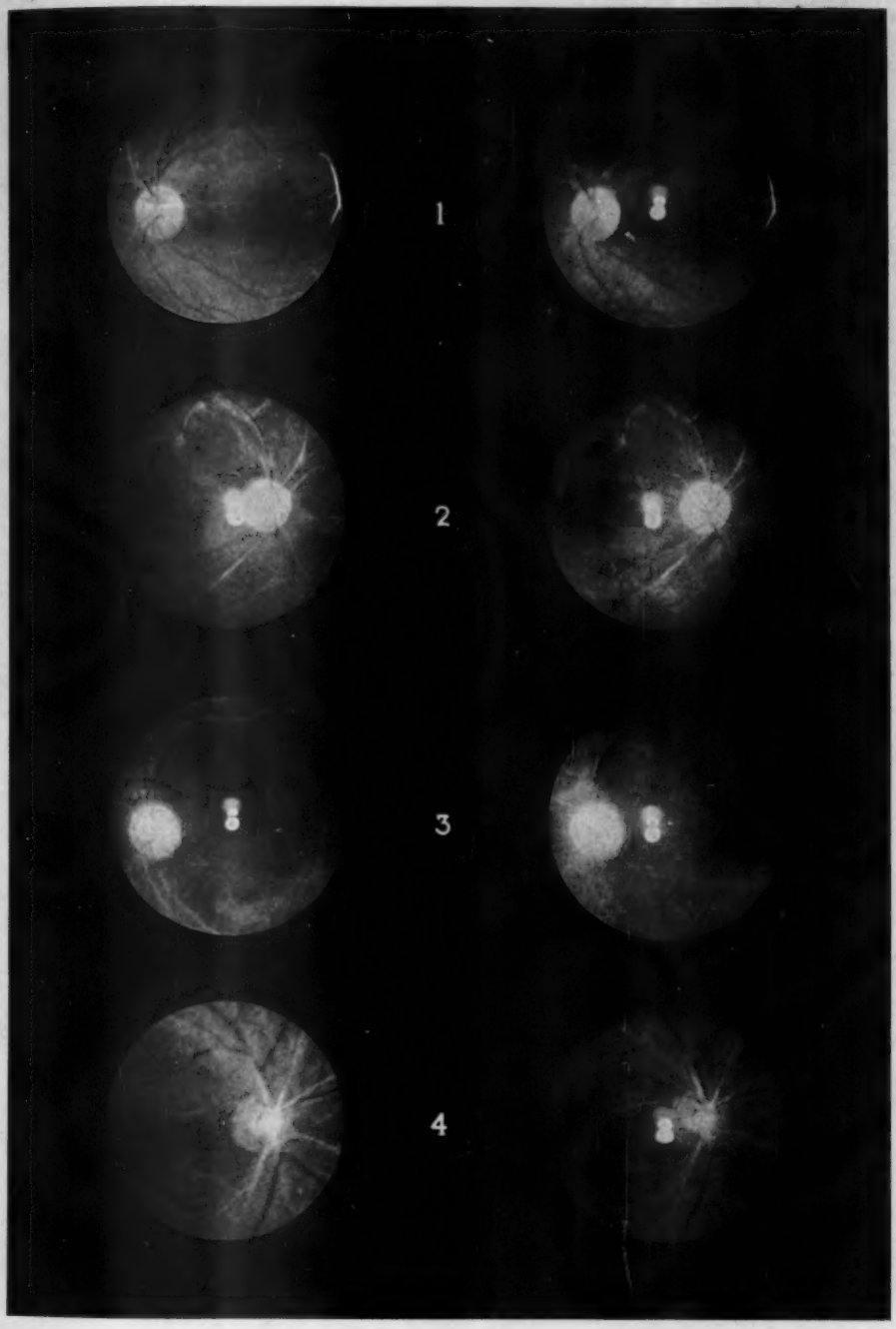



Fig. 7.—Arterial sclerosis.

On March 26, the hemoglobin was 100 per cent and the red blood cell count was 5,740,000. There was free hydrochloric acid.

The Wassermann reaction was negative.

This improvement followed the ingestion of liver. The entire arterial tree (fig. 7, *4*) was a white fibrous structure with a thick rootlike mass over the center of the disk. The size of the arteries and the occasional dark line showed that they were patent. The veins were about normal in size and distribution.

Another example of the different appearance of the fundi in the same patient is presented in figure 8.

CASE 16.—A woman, aged 62, single, had noticed a slight change in the vision of the left eye for three and a half years before examination, and for the past three days had been worried because of a spot before her right eye. The systolic blood pressure was 168. Vision was 20/70.

There was general retinal edema (fig. 8, *1*) with large, full, tortuous veins showing wide bands at the artery crossings. There was an increased wall reflex on all of the vessels, even on the narrow arteries. The dark macula was speckled with a few retinal blood dots.

In the other eye (fig. 8, *2*), vision was 20/200. A gray edematous disk and retina were present. The veins were tortuous but not as full as in the right eye. The arteries showed changes ranging from stiff walls to white sheaths and to enlarged chalk-white tubes. There were many twistings in the small new capillaries between the disk and the macula, and several exudate flecks. A dark granular ring was seen in the central region around the fovea.

CASE 17.—A woman, aged 55, had recently noticed dimness in the vision of the left eye; it was found to be 20/50 and improved to 20/30. Her systolic pressure was 185 and she had no constitutional symptoms.

The temporal view of the eye (fig. 8, *3*) showed white vessels, an irregularly pigmented macula and fine capillaries ending in globular aneurysmal dots, occasionally surrounded by exudate.

The nasal half of the fundus was edematous (fig. 8, *4*), so that the disk was thick and clouded. The arteries compressed the veins where they crossed them. A region of completely white tubular vessels and partially changed ones was confined to an area near the temporal macula, where the fine, twisted lines were best seen with small blood dots at the end of the vessel. These were advanced pipestem arteries, which develop in women who have not been exposed to severe muscle strains.

CASE 18.—A married woman, aged 55, first came under observation two years before the present writing, because vision in the left eye had fallen to 1/200. The systolic pressure was 142; there were considerable sugar in the urine and a high blood sugar content. This case furnished the best illustration of perivascular lymph sheath infiltration that I have ever seen.

The left eye (fig. 9, *1*) had advanced vessel changes. A conical capillary tangle and a less striking vessel formation were seen in front of the disk. The distance from the anterior retina to the choroid was demonstrable, for the deep, dead white exudate was beneath the vessels, many of which were partly covered by a similar exudate. The perivascular streaking was extreme.

The lower quadrant of the same eye (fig. 9, *2*) presented the new vessels extending into the vitreous, and the exudate as isolated dots, masses and ensheathing tubes. At this time the patient had a small amount of albumin but no sugar in the urine. The blood sugar was 102; the basal metabolism was + 19.

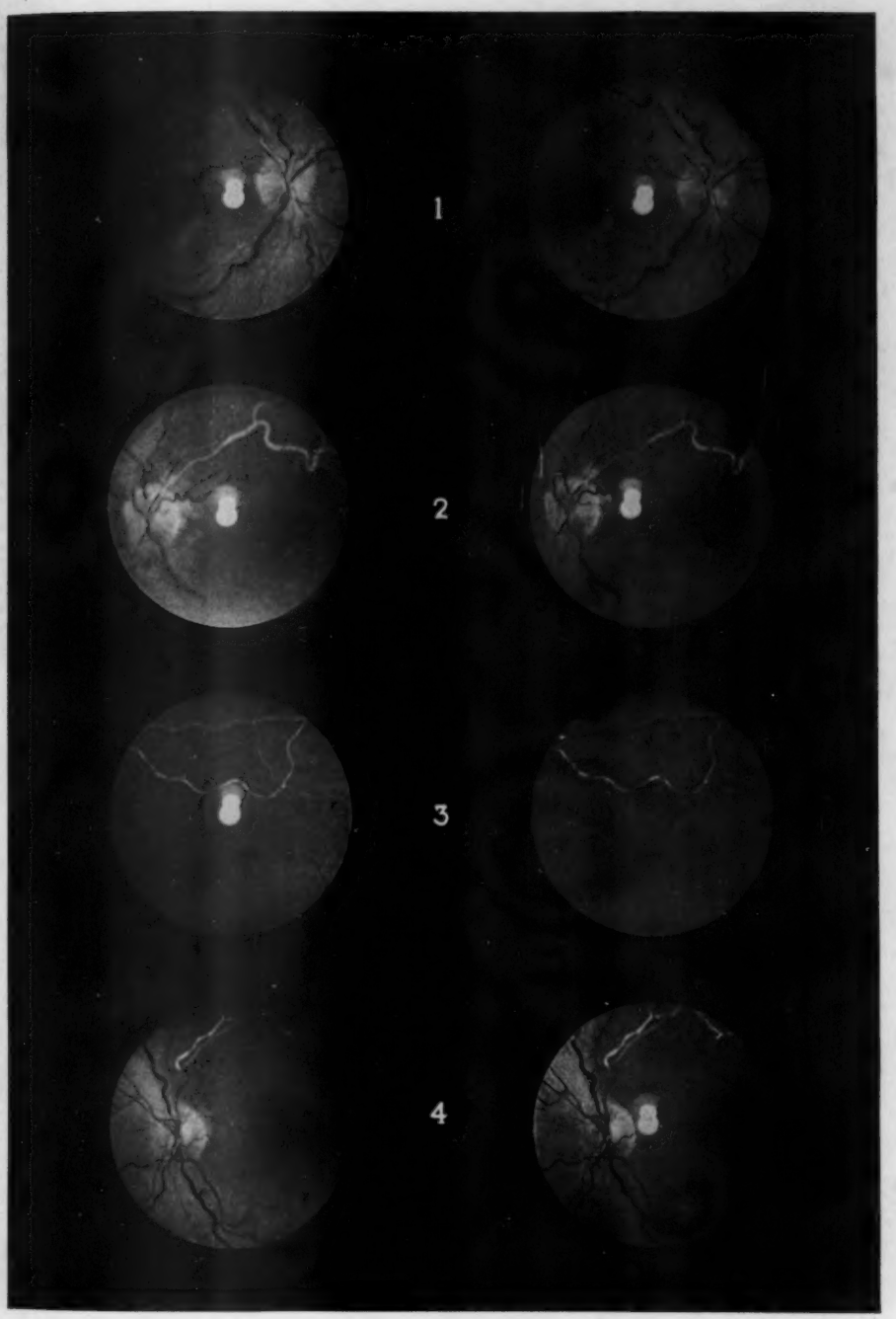



Fig. 8.—Atheromatous arteriosclerosis.

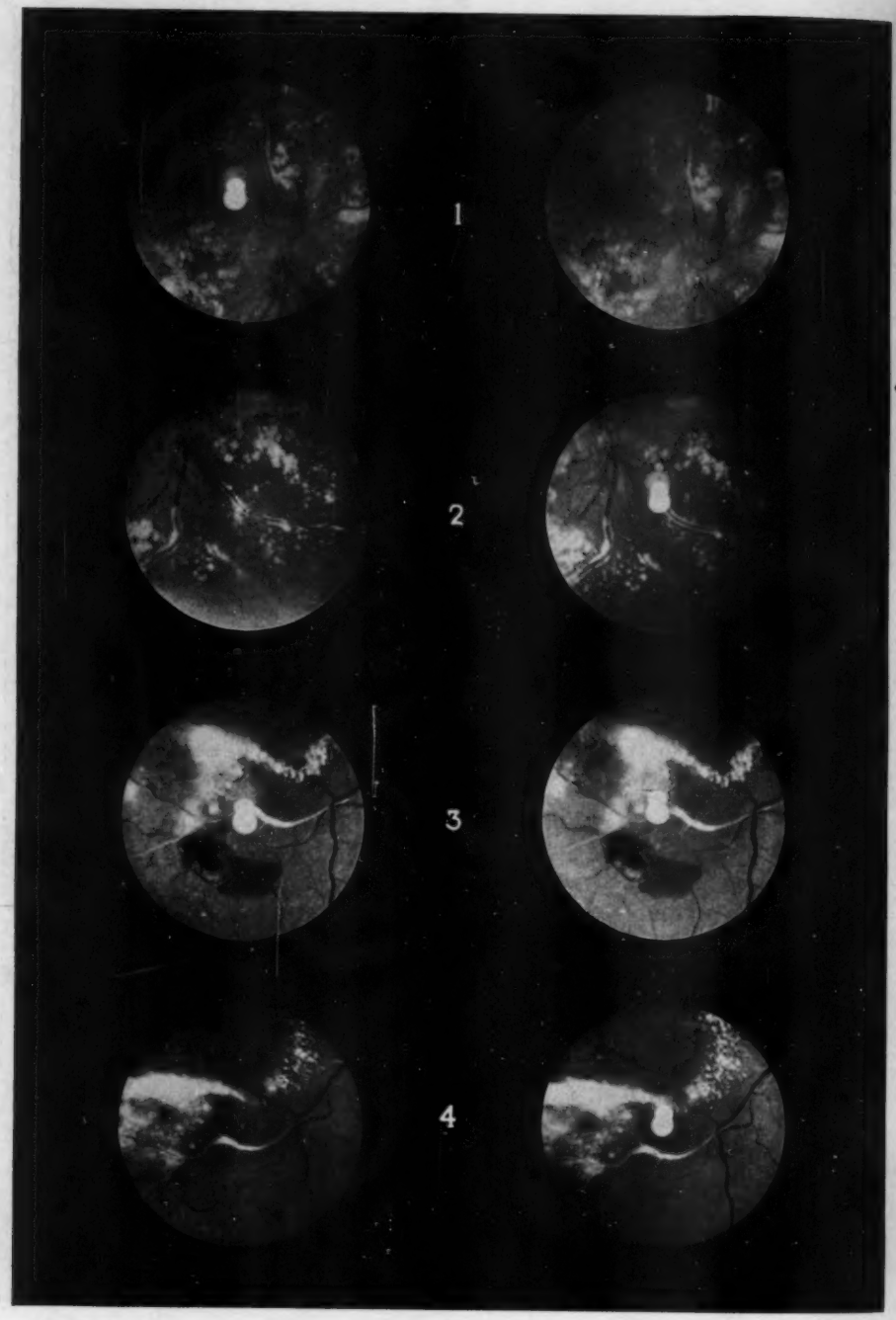



Fig. 9.—Syphilitic periarteritis and sclerosis.

CASE 19.—A man, aged 49, had had syphilis for ten years. Vision was 20/200. The illustration shown (fig. 9, *3*) is of the lower half of the fundus, with the dark macula at the upper border of the picture, a flocculent, bright, whitish-yellow circinate exudate and a smudged white homogeneous mass. The atheromatous artery could be traced from a normal caliber central reflex portion to a vein; when the vessel passed beyond this it was at first a thin, white line but soon widened into a bright white band which bifurcated farther on. The periarteritis was visible in the branches. The granular hemorrhage was retinal and outlined by a vein which was in part covered by the blood. New retinal capillaries were found between the atheromatous vessel and the circinate-like exudate.

The absorption of the hemorrhage, the increase in the flocculent exudate and the transition of the homogeneous mass into the speckled type were striking changes (fig. 9, *4*). The white-walled artery was a little smaller. In the macular zone, seen through the transparent retina, white arterioles and a dark ring of pigment were found. The fusiform beading in the artery near the vein on the right side of the photograph is interesting.

### HYPERTENSION

If hypertension is considered as a condition in which there is an elevated blood pressure without other demonstrable physical observations, I believe that it is not possible in the early case, or even in one that has persisted for a long time, to make a positive diagnosis of hyperpiesia, since hypertension may or may not produce visible changes in the fundus vessels. The degree of pressure does not seem to be the essential factor in the production of the fundus disease.

Hypertension sometimes shows itself as an increased twisting of the smaller arteries as they extend from the disk toward and around the macula. This appearance is suggestive, but it is by no means a constant observation; in fact, my own cases seem to suggest that it is infrequent. The corkscrew twists develop without signs of vessel compression and it is only later in the course of the disease that the increased visibility of the artery wall and the usual arteriosclerotic changes develop.

The fundus picture that most often suggests hypertension is one in which the disk is congested by the increased fulness and visibility of the nerve head capillaries. The disk frequently appears pink or even red. In a certain percentage of these patients there is associated with the hyperemia some edema, a few hemorrhagic spots and an occasional exudate. As the disease progresses these symptoms increase, and a few glistening punctate exudates may appear near the macular region. Before these alterations have become well established, arteriosclerosis of the retinal vessels becomes a prominent factor. It is sometimes impossible to distinguish the true arteriosclerotic atrophy from the final stage of hypertension. I wonder whether they are not identical.

Recent literature is replete with many illustrations and several word pictures of the condition called hypertension. In fact, the retinal appearances have been divided into four stages. Photographically, it

does not seem possible to demonstrate any clear line of demarcation between these periods, nor am I sure that hypertension is more than an accompanying symptom or perhaps a causative agent which may in turn be caused by another common factor.

For fear that this statement will be misunderstood, attention is called to figure 10. The illustrations presented are typical of the condition called hypertension. They are so distinctive that there can be no doubt that they are at least concomitant with the long continued increased blood pressure. But even here one must sound a note of warning, for it is not impossible to believe that sensitive tests for kidney function may be inefficient in the determination of early kidney dysfunction. If this then is a typical hypertensive fundus, is it not somewhat surprising to find that practically the only point of difference between it and some cases of frank chronic nephritis seems to be a degree of retinal edema?

Figure 10 may make this statement a little clearer, for in it may be seen the foveal exudates, the line of deep perimacular exudate and superficial hemorrhages and compression of the veins—everything except edema, and a pressure lower than in the first case.

Fortunately the same picture shows the retinitis that comes after long-continued hypertension and the type that is seemingly primarily nephritic. Study of the illustrations will do more than lengthy descriptions to separate the two conditions, or at least the two stages of what may eventually be proved to be the same disease. The reason why some patients can live for decades with high blood pressure and never show anything but arteriosclerotic changes in the fundi is still enfolded in stygian darkness.

CASE 20.—A woman, aged 48, had been under observation for twenty years. Seven years before the present writing, her blood pressure was 240 systolic and 110 diastolic. She showed no evidence of fundal change until the past year. Then an indefinite headache developed. Her blood pressure at that time was 290 systolic and 150 diastolic. Physical examination and kidney function and laboratory tests did not show anything except the hypertension. In the right eye (fig. 10, *1*), vision was 20/40. As the edema subsided the retinal striations and large patches of snow exudate became visible, with thin smoke remnants over the superficial vessels. The veins and arteries were tortuous, the former with angular kinks and the latter with undulations. Although the process was far advanced, there was a slight vein compression with no streaks of periarteritis. There was, however, a great change in the macular region, which was swollen, darker than normal and partially concealed by the cloudy edema of the overlying vitreous. The lower border of this edematous region presents several denser spots which could be easily distinguished from the deep, white dots found close to the fovea, the latter constituting the points of a beginning macular star.

In the left eye (fig. 10, *2*), vision was 20/30. The cloudy swelling of the retina hid some detail, but it was a favorable field in which to observe the kinking and curving of the arteries, the former as the vessels dipped down,

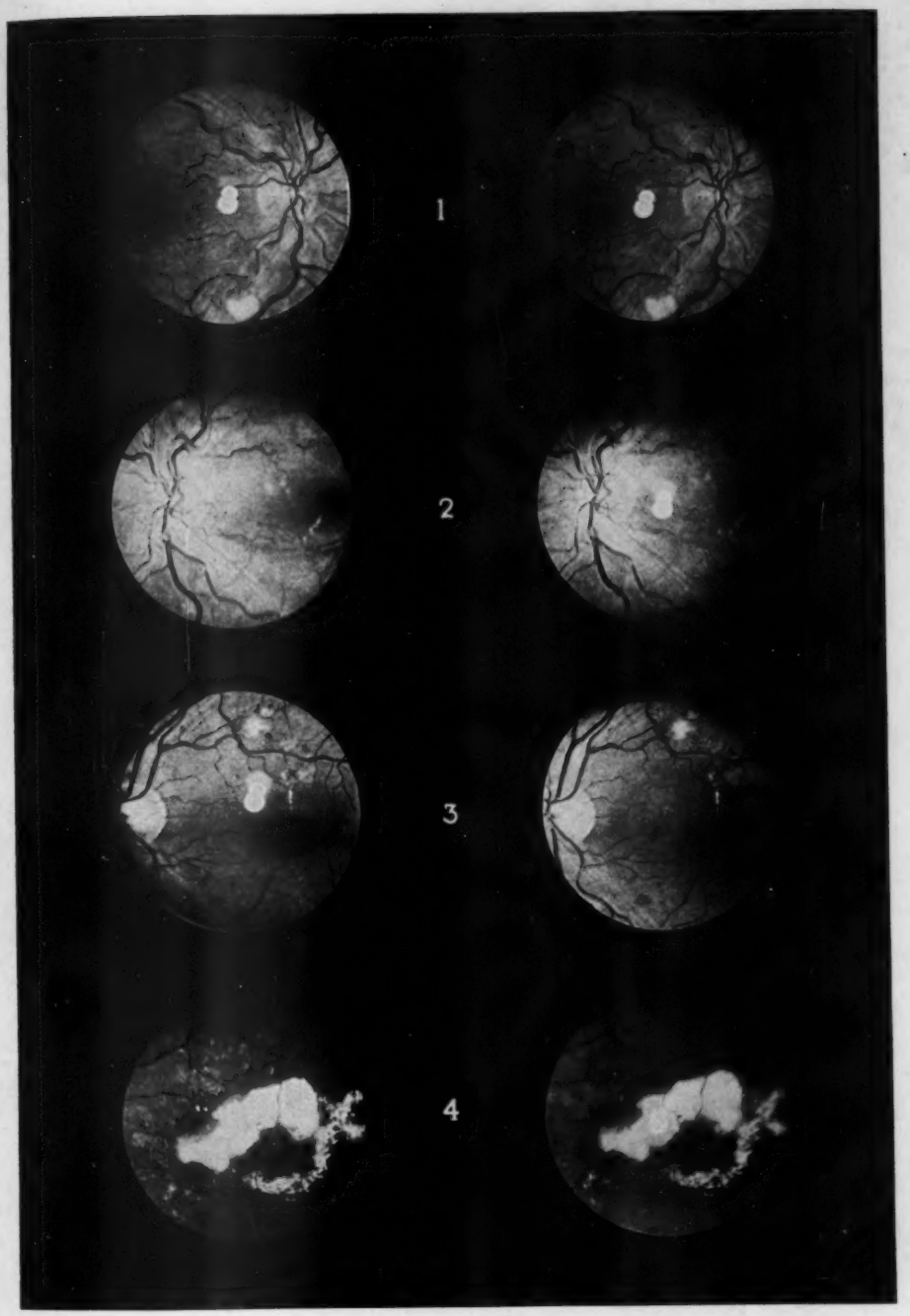



Fig. 10.—Hypertensive sclerosis and nephritis.

the latter on the surface. No arterial sclerosis was noticeable except below the macula. The illustration shows an interrupted capillary stream near the macula, where the superficial, fuzzy white exudate and the deeper, well outlined macular star line can be seen.

CASE 21.—A man, aged 54, had a blood pressure of 160, with albumin and sugar in the urine. Vision was 20/40.

There was only a faint change in the arteries (fig. 10, *3*), and yet several balls of blood and a few gray to white fluffy exudates of absorbing edema were observed. One deep, yellowish-white line of exudate extended upward from the macula.

CASE 22.—A woman, aged 59, had a blood pressure of 192. Vision was 1/200.

The composite perimacular photograph (fig. 10, *4*) shows a large, homogeneous white mass of exudate and a crescent of white flocculent deposits, retinitis circinata. The white surface makes it possible to bring the fine capillaries into relief and also to show a clot with its surrounding ring.

The lesser degrees of hypertensive change are built around the edema of the disk. The symptoms of edema of the disk is one that must be given brief attention. The difference in a level between the retina and the choroid is variable. In a large class of cases it is impossible to state where physiologic elevation becomes pathologic swelling. If one is to base a diagnosis on the haziness of disk outline, he is confronted with the innumerable differences in the thickness of the nerve head. If one is to consider edema as the loss of transparency, he must understand the appearance of the fundus in frank nephritis, and the hypertensive and pure arteriosclerotic conditions. This seems rather difficult, and the more I study the problem the more I am inclined toward Mr. Foster Moore's classification into arteriosclerosis and arteriosclerotic retinitis.

Perhaps nothing can be said that will bring the proponents of each theory to the battle line more determined to defend their chosen theory than the statement that arteriosclerosis may explain all of the fundal changes, whereas hypertension can account for them only if it is said that the hypertension produces a sclerosis.

The illustrations must speak for themselves. My deductions are made from the analysis of 9,000 photographs. I expect them to stimulate investigation and perhaps clear the way for a more generalized study of the problems surrounding the alterations that take place in the blood vessels.

## SUMMARY

The appearance of the blood vessel walls affords an excellent insight into the past, an explanation of the present and a glimpse into the future. The possible symptom differentiating embolism of the central retinal artery from thrombosis of the same vessel is the extravasation of blood about the nerve head. Arteriosclerosis is a clinical pathologic process in which the only change may be in the blood vessels. Arteriosclerotic retinitis is an inflammatory retinal change added to a persistent arterio-

sclerosis and is probably one of the terminations of the sclerosis. Renal retinitis is characterized by hemorrhages, exudates and blood vessel changes. The vogue of ascribing to high blood pressure almost every symptom or complaint has led too many away from arterial degeneration. Hypertension may or may not be an entity. In some cases of diabetes the retinal vessels are outlined by a yellowish-white exudate. The vessel changes in syphilis are a particular subdivision of arteriosclerosis. The fundal arteries are smaller in retinitis pigmentosa than in syphilitic retinochoroiditis of the same stage of pigmentation. Lines of migrated pigment along the retinal vessels are either from retinitis pigmentosa or from syphilis.

# SMALL TUMOR OF THE IRIS WHICH CAUSED SECONDARY GLAUCOMA

## RELATION OF MELANOMA TO SARCOMA OF THE IRIS *

ARNOLD KNAPP, M.D.
NEW YORK

W. L., a man, aged 30, seen on Feb. 24, 1928, stated that the sight in the right eye had become affected six weeks before, and that the eye had felt uncomfortable. Vision with the correcting glass (—0.5 sphere, —0.5 cylinder, axis 45 degrees) was 6/9. The anterior chamber was deep. The pupil measured 8 mm. and was immobile. Tension, as measured by Schiötz' tonometer, was 42. The field was concentrically contracted. The optic nerve showed a deep cup. The iris was blue, except for a sector between 3 and 5 o'clock, which was brown. This sector was perfectly flat, though suggesting some thickening at the periphery. Though one eye had always been darker than the other, the patient's mother had noticed the spot only nine years before. The left eye was normal.

The most careful examination of the iris did not confirm the presence of a tumor that might be the cause of the glaucoma, and on May 6 a trephining was done above. The tension was relieved.

The patient was not seen again for one year. On May 3, 1929, the vision was found reduced to 20/70. Tension was normal; there was a cystoid scar. The discolored area in the iris presented a distinct brownish mass in its outer half which sprang from the surface of the iris and extended into the chamber angle. This mass measured 4 by 1.5 mm. A general physical examination was made and yielded negative results. The urine did not give a melanin reaction.

As there was now no question of the presence of a growth, enucleation was advised and performed on June 4, 1929.

The eye was sectioned after hardening and was embedded in paraffin in Dr. Jonas Friedenwald's laboratory. Microscopic examination showed the following condition:

On one side the iris was somewhat thickened and filled with a brownish mass which extended and invaded the iris root and the adjacent sclera and pectinate ligament (fig. 1). This swelling consisted of a tumor in which three layers could be recognized: One was superficial and composed of compact spindle cells projecting into the anterior chamber with a wavy, uneven contour; it was of varying pigment content and was continuous with the similar process in the angle and along the scleral spur. The region of the pectinate ligament was packed with pigment-bearing cells, and this infiltration extended well into the sclera. The chamber angle was open. Underneath was the second layer, which was more closely pigmented; the cells were less crowded together, the nuclei were round and less elongated and their direction was downward into the iris stroma (fig. 2). The third layer occupied the iris stroma, which had taken on a deep eosin stain and was packed with cells. These cells were less pigmented and possessed large

* Submitted for publication, June 3, 1930.

* Read at the Meeting of the American Ophthalmological Society, Hot Springs, Ark., June 2, 1930.

nuclei. There were many vessels with thickened hyaline walls, and the immediately surrounding cells appeared especially like endothelium (fig. 3). In the peripheric sections where the infiltration of the iris stroma was less pronounced, the resemblance of these cells to the connective tissue cells (chromatophores) of the iris stroma was apparent, and the branching protoplasmic processes were broader and had taken on a deeper eosin stain. Some of the vessels contained pigmented cells in their walls and in the lumen. The pigment epithelium on the posterior surface of the iris showed no change.

In the sections adjoining the growth definite sarcomatous cell groups were visible like implantation metastases on the anterior surface of the iris; the infiltration in the sclera was pronounced.

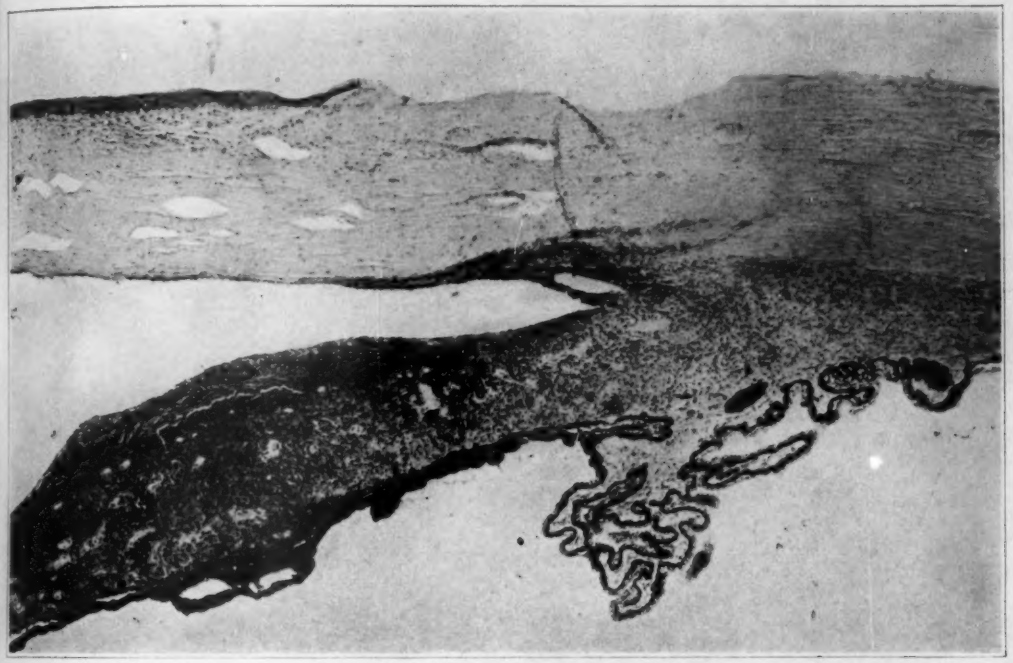


Fig. 1.—Superficial distribution of the spindle cell part of the tumor, with extension along the open chamber angle to include the region of Schlemm's canal and adjoining sclera; reduced from a magnification of ×75.

The iris on the opposite side showed accumulations of deeply pigmented spindle cells just beneath the anterior margin. The pectinate ligament contained pigment cells.

At the limbus there was a gap, the site of the trephine operation, which was partly lined with endothelium, and at one side a thin strip of iris was drawn into the gap for a short distance. This area was covered by loose edematous connective tissue, and the overlying epithelium was intact.

The eye otherwise showed no change except a depressed cribriform plate and optic atrophy.

Pigmented nevi or, more properly, melanomas of the iris are always regarded with suspicion, as they may give rise to malignant tumors. It is well to remember that it is the pigmentation of the anterior layers of the iris that gives it its darker color. This pigmentation is the physi-

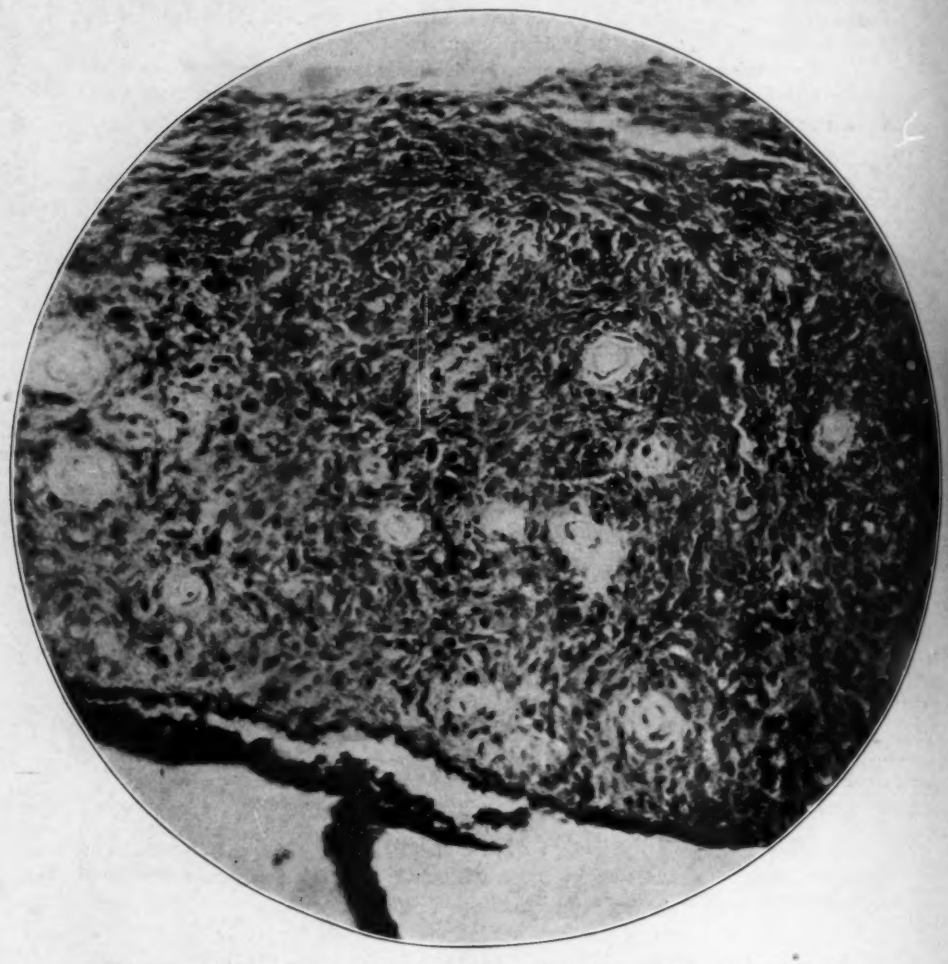


Fig. 2.—Topography of the tumor, and its division into three layers; × 200.

ologic distribution of pigment cells which correspond to the chromatophores of the stroma and do not represent an abnormal tissue (Morax [1]). Wolfrum [2] expressed his belief that the melanomas are a form of intermediate stage between normal tissue and malignant neoplastic processes.

1. Morax: Cancer de l'appareil visuel, Paris, Gaston Doin, 1929, p. 229.
2. Wolfrum: Graefe's Arch. f. Ophth. **71**:195, 1909.

Collins [3] divided melanomas of the iris into: (1) hyperplasia of the surface cells of the iris, (2) groups of deeply pigmented cells, "clump" cells situated in the iris stroma, (3) those arising from the pigment

Fig. 3.—Character of the endothelium-like cells in the iris stroma which resemble chromatophores; pigmentation and invasion of the thick and vascular walls; reduced from a magnification of × 500.

epithelium on the back of the iris and (4) general melanomas in association with a general melanosis of the eyeball. He examined four cases of

3. Collins, in discussion on Melanomata, Tr. Ophth. Soc. Kingdom **46**:98, 1926.

malignant melanotic growths in which innocent melanomas had been noted in the iris. The innocent formation consisted of a hyperphasia of the surface endothelium of the iris and the formation of "clump" cells, and the malignant growths were of the nature of spindle cell sarcomas. He took for granted the endothelial origin of the melanomas of the iris and that the metaplasia of endothelial cells into spindle-shaped cells is a well known phenomenon in the tissues in the interior of the eye.

Watzold,[4] however, said that Wolfrum had definitely proved that there is no endothelial membrane on the anterior surface of the iris.

According to Sattler,[5] tumors may originate in rust brown or dark brown spots in the iris. These spots consist in a thick conglomerate of chromatophores in the anterior layer of the iris, and in these places the iris is somewhat thickened. The cells are often plumper and more pigmented than the other chromatophores, and in addition unstained branching stroma cells are sometimes seen. The pigment granules are unevenly large and hide the nucleus. The limitation from the surrounding tissue is not definite. Melanomas may occur in blue irides with little pigment and may remain unchanged through life. When there are a number of pigment spots it is only one of them that becomes transformed into a malignant growth. Curiously enough, sarcoma of the iris has usually been observed in the lower half of the iris. The growth at first is very slow and without any symptoms. As the tumor extends to the root of the iris it involves the chamber angle and the anterior part of the ciliary body; then glaucoma develops. When the tumor has reached the angle, tumor cells can be observed passing through the pectinate ligament to Schlemm's canal, then extending along the vascular channels to the anterior ciliary veins. Iris sarcoma generally consist of spindle cells crowded together in bundles interlacing one another in different directions. Sometimes round cells are also observed. Sattler expressed his belief that the so-called clump cells have nothing to do with the development of the sarcoma. In the nonpigmented parts of the tumor there are usually round and polyhedral cells with round or oval nuclei. As they are frequently arranged around vessels, they have been described as perithelium. Sattler, however, was not in favor of the view that the cells in a leukosarcoma originate from endothelium.

Mayou[6] stated that the "large spindle cells are characteristic of growths derived from naevi." A feature of the growth is the rapid involvement of the pectinate ligament, and that secondary glaucoma

4. Watzold: Tumoren der iris, in Lubarsch-Ostertag Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie des Menschen und Tiere, Wiesbaden, 1913-1925, part 1, p. 292.

5. Sattler: Die bösartigen Geschwülste des Augen, Leipzig, Herzel, 1929, p. 54.

6. Mayou: Sarcoma of the Iris, Brit. J. Ophth. **14**:152 (April) 1930.

follows. Increase in the size of the tumor defines its malignancy. This author spoke of large cells of endothelial type, such as are usually found in nevi, together with large quantities of pigment cells. Unfortunately, these characteristics are not shown clearly in his illustration.

Considerable confusion has been introduced into the subject of tumors of the iris, and the term nevus is particularly misleading. It is certain that melanosarcomas are derived from stroma cells and have nothing to do with true nevi.

In the tumor described in this paper, the superficial part is the usual spindle cell melanosarcoma. The deeper parts situated in the iris stroma are curious on account of the large nuclei with diffusely stained protoplasm and hyalinized vessels. These cells might suggest an endothelial origin and that the malignant melanosarcomatous tumor was invading this preformed tissue. In peripheral sections where the infiltration does not fill the iris and in depigmented sections the similarity of the cells can be traced, and the entire picture is that of a tumor composed of one type of cell.

A similar tumor was described by Raubitschek [7] from the Elschnig clinic. It was brownish gray and approached the angle of the anterior chamber. The tumor and adjoining ciliary body were removed at operation. Microscopic examination showed a normal posterior iris and many vessels with thickened hyalinized adventitia. The mass could be subdivided into several formations. The one at the anterior surface was the most definite. This consisted of fusiform cells with long cell bodies thinning to each end. The nuclei were oval, and stained with hematoxylin, showing nuclei and a chromatin framework. The cells were crowded together. In brief, there was a closely packed cellular tissue arranged in parallel rows, and bands of circular cells and cell nuclei which were the same tissue only cut at right angles. This region was without pigment except at the periphery, where it approached the pigmented area. The rest of the tumor was darker and of netlike structure. At the root of the iris there was another formation consisting of small, irregular, polygonal cells with prolongations, dark-stained nuclei and moderate pigment. In between these was a layer of elongated pigmented cells with indistinct cell margins, and crowded together a collection of narrow dark stained nuclei. The interior of the tumor consisted of an irregular network of elongated clump cells with swollen nuclei, not deeply stained, and an indistinct chromatin structure. This area was thickly pigmented. In short, the tumor consisted of an unpigmented part resembling a spindle cell sarcoma, and a tissue of polygonal pigmented elements suggesting an endothelioma.

7. Raubitschek: Ueber Iris Tumoren, Klin. Monatsbl. f. Augenh. **52**:683, 1914.

Ribbert, however, said that a tumor must arise from a single chromatophoroma passing through several unpigmented stages, so that melanosarcomas show areas without pigment consisting of round, spindle and epithelioid cells. Schieck was of the same opinion and regarded all cells, pigmented or nonpigmented, in these tumors to be derived from chromatophores.

Heine [8] described a case in a woman, aged 35, whose grayish blue iris showed a small brown melanoma which had existed for years but which had increased in size recently. Microscopic examination showed that half of the tumor was a melanoma which gradually passed into the other half, a spindle cell sarcoma.

The curious clinical feature in my case was that the pigment spot did not seem prominent enough to warrant the diagnosis of a tumor, though it was large enough to produce glaucoma; the presence of the glaucoma should have been sufficient to make the diagnosis. One year later a growth had definitely formed, and the eye was removed.

Clinically, the development of a malignant tumor in the iris from a melanoma is characterized by the increased growth of the process on the surface of the iris, by a probable change in color of the newly formed tumor from that of the original melanoma and by the encroachment and invasion of the chamber angle with the early development of glaucoma.

---

8. Heine: Zur Melanose und Sarcose der Iris, Graefe's Arch. f. Ophth. **113**: 417, 1924.

# Correspondence

## NOTES FROM VIENNA

*To the Editor.*—One meets with diversity of opinion here, as elsewhere. In the Lindner Clinic the trephine and often cyclodialysis are resorted to in the treatment for glaucoma, whereas in the Meller Clinic iridotasis or iridencleisis is the favorite procedure.

All cataracts, with the exception of the hypermature forms, are removed intracapsularly, modifications of the Kalt forceps being employed. Peripheral iridectomy is performed in every case and always after delivery of the lens. The wound is stitched tightly with three conjunctival sutures, and the patient is allowed to walk back to the ward and remain up. Complications are rare. Blocking of the orbicularis muscle is practiced in the Meller Clinic. Retrobulbar injections are not employed for cataract operations in either clinic, whereas in Professor Meessmann's Clinic in Berlin it is employed in every case.

Bloodletting is practiced immediately before operation in patients with glaucoma and high blood pressure.

Tebeprotin is employed in Meller's Clinic in the treatment for tuberculous diseases of the eye, and excellent results are reported by Dr. Urbanek under whose guidance it is given.

A large number of detached retinas are operated on by Gonin's method, and excellent results are obtained in the Lindner Clinic, owing to the accurate localization of the tears in the retina by the ingenious method devised by Dr. Guist. It requires a great deal of patience to localize the tears by his machine, but so far it is the only means of doing this accurately. It is a simple matter to perform the cautery puncture, but localizing the holes is difficult, and, unless done exactly, spells failure. In the Meller Clinic the refractometer is employed for localizing, but this, like Gonin's method, is not exact, and thus many failures and near failures are caused.

The machine of Dr. Guist consists of a combination of the Gullstrand lamp and perimeter. The hole or holes are located and read off on the perimeter. A small metal shell is graduated in degrees to 180 on its circumference, and to this is attached one or more thin pliable brass arms, according to whether there is one or more holes. Each arm is cut so that the distal end will be just in front of the hole, and it is placed on the meridian of the shell corresponding to the meridian on the perimeter at which the hole is located. The shell is sutured over the cornea, and the conjunctiva is dissected up where the puncture is to be made. The sclera is lightly touched with the cautery point at the end of the arm to mark the location of the hole, and the shell is then removed from the cornea. With the cautery point at white heat the sclera is punctured and the subretinal fluid allowed to drain; then the point, at red heat, is reintroduced for a distance of 4 mm. and allowed to remain for from five to ten seconds.

In all operations on the globe, with few exceptions, the eyeball is held immovable by means of a stitch in the superior rectus or at times in the inferior rectus. The ingenious speculum of Guist, which is really a modification of another speculum, obviates the necessity of an assistant to hold this stitch. On the upper and lower lips of the speculum is a small hole by which the rectus muscle is stitched to the speculum and holds the eye absolutely immovable without tension. At either end of the speculum is a small support which, when screwed up, holds the lids away from the globe.

T. H. Odeneal, M.D., Beverly, Mass.

---

# News and Notes

Edited by Dr. John Herbert Waite

## SOCIETY NEWS

**National Society for the Prevention of Blindness.**—The society has announced its 1930 Annual Conference to be held November 17 and 18 at the Russell Sage Building in New York City. The program includes conferences on: The Conservation of Vision in School Children, the Relation of Lighting to Vision Conservation, Sight-Saving Classes and Medical Association Services.

**American Academy of Ophthalmology and Otolaryngology.**—The thirty-fifth annual meeting of the American Academy of Ophthalmology and Otolaryngology was held at the Hotel Sherman, Chicago, Oct 27 to 31, 1930, under the presidency of Dr. William H. Wilder of Chicago. The guest of honor was Prof. Dr. Emile de Grosz of Budapest, Hungary. The following officers were elected for the coming year: President, Dr. John F. Barnhill, Indianapolis; president-elect, Dr. S. Hanford McKee, Montreal, Canada; first vice-president, Dr. Meyer Wiener, St. Louis; second vice-president, Dr. J. W. Carmarck, Indianapolis; third vice-president, Dr. Ben Witt Key, New York; comptroller, Dr. Secord H. Large, Cleveland; executive secretary and treasurer, Dr. W. P. Wherry, Omaha; secretary, section on ophthalmology, Dr. William P. Benedict, Rochester, Minn.; secretary, section on otolaryngology, Dr. John H. Meyers, Kansas City, Mo.; secretary, section on instruction, Dr. Harry S. Gradle, Chicago; assistant secretary, section on instruction, Dr. William V. Mullin, Cleveland; editor, transactions, Dr. Arthur Proetz, St. Louis; assistant editor, Dr. Lawrence Post, St. Louis; member of council, Dr. Ralph A. Fenton, Portland, Ore., and member of board for ophthalmic examinations, Dr. William H. Wilder, Chicago (to succeed Dr. James M. Patton). The place of meeting for 1931 is French Lick, Ind.

**American Ophthalmological Society.**—The society will hold its 1931 meeting either in Asheville, N. C., or in Swampscott, Mass.

# Obituaries

## THEODOR AXENFELD, M.D.
1867-1930

In the death of Theodor Axenfeld, on July 29, 1930, ophthalmology, not of Germany alone, but of the world, suffered a serious loss. Born in 1867, in Asia Minor, the son of a German pastor, he was brought back to his family's home in Godesberg a few years later. Entering the neighboring University of Bonn at the age of 17, he passed his state examination when he was 22. His first introduction into ophthalmology was through Schmidt-Rimpler. In 1894, he was in Marburg, attached to the master Uhthoff, who even at that time recognized the great promise of this young man, and throughout life remained his true friend and kindly adviser. Probably there was no ophthalmologist during Axenfeld's career whom he looked up to as he did to Uhthoff, his early chief. Many were the nights in Marburg that Axenfeld burned the midnight oil while engaged in his studies. He did not hold himself entirely aloof from social gatherings, however, to which his love of music, his fine voice and his simple and open address made him a welcome visitor.

In 1895, Axenfeld became privatdocent, and accompanied Uhthoff to the University of Breslau. In 1897, at the age of 30, his talents and promise were sufficient to secure for him the position of full professor and director of the ophthalmologic department in Rostock. He was one of the youngest at that time to attain such a rank. In 1901, he was called to the chair at Freiburg to succeed Wilhelm Manz, and at that university he spent the remaining twenty-nine years of his life. Here he built up what might almost be called a school of ophthalmology, to which students flocked from all parts of the earth; here he edited and built up the *Klinische Monatsblätter für Augenheilkunde;* here he pursued many of his scientific investigations and wrote many of the works which made his name well known throughout the world. The clinic in the Albertstrasse became a mecca for students and visitors, and few came who did not take away with them something of real value.

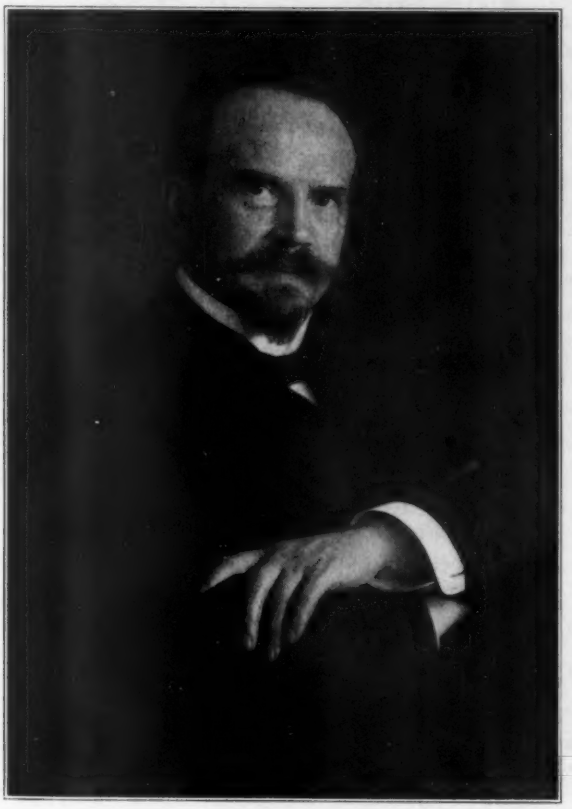
The greatness of Axenfeld lay in the simplicity of his nature, his great love of truth and his love for and service to humanity. His Christian interest in humanity could not tolerate or understand the international hatred of the World War. He combined in himself the best qualities of the investigator, teacher, organizer, editor and prac-

ticing physician; in truth, he was a many-sided man. Which of these qualities we should rate highest, it is hard to say. As a lecturer and teacher, Axenfeld was without a peer; his language was simple, his delivery eloquent and compelling. He was stimulating, helpful, always full of new ideas and with interesting views on all questions. He had a masterful command of ophthalmologic literature.

In 1902 and 1903, I spent eight profitable and happy months in his clinic. At that time the student group consisted of men from Germany, Austria, Italy, Russia, Japan, Canada and the United States. It was truly a happy family, with the young professor as its head. He took a personal interest in each man. If defects showed themselves in the medical training of any of us, we were sent to the appropriate department to take a course and remedy them. The laboratory work was stressed; each man was stimulated to an "Arbeit," large or small, his results were checked up from time to time, and his knowledge of the literature was tested. Finally, he had the supreme pleasure of seeing his work, if worthy, appear in the *Monatsblätter*. The day was long. The undergraduate students of the university came in for their instruction at a quarter past 7 in the morning; at this exercise most of the "Voluntär-Assistents" helped. This was followed by the lecture, treatment of the patients in the polyclinic, operations, lunch and, in the afternoon, laboratory work. Once a week the professor held his "Referierabend" at his apartment, presided over at that time by his charming sister. Many were the delightful evenings spent there, either purely social or in reviewing the literature and discussing scientific subjects. The professor was the despair of his sister; he rose early, paid little attention to meals, and many a night his light could be seen burning till long after midnight.

Axenfeld's own investigations embrace almost every branch of ophthalmology, but he leaned rather to the laboratory than to the clinical side. His "Habilitationsschrift" on purulent metastatic ophthalmia, published in 1895, was awarded the Graefe prize of the Heidelberg Society. His work with Uhthoff on the relation of the pneumococcus to serpent ulcer, and his discovery coincidentally with Morax of the diplobacillus which now bears both their names, were outstanding achievements, as was also the proof of the identity of the xerosis bacillus with the so-called pseudodiphtheria organisms. In 1904, appeared his "Bakteriologie in der Augenheilkunde," of which a new edition has recently been published. Later came his monographs on trachoma, vernal conjunctivitis and many other subjects. The work with Stock on the importance of tuberculosis in the etiology of intra-ocular hemorrhage, the so-called periphlebitis tuberculosa retinae, brought to attention a new point of view. Many investigations inspired by him

appeared from the pens of his students. His textbook and atlas, which has passed through seven editions, must be rated among the best books of its kind. His enormous industry was at once the admiration and despair of his colleagues and pupils. How he could squeeze extra precious minutes out of the twenty-four hour day was to him always one of his most pressing problems. For thirty years, he edited the *Klinische Monatsblätter,* which alone might be considered a full-time



THEODOR AXENFELD
1867-1930

task, and raised it to the position of the best and the most widely read of the world's journals on ophthalmology. For many years he worked on it with practically no clerical assistance. His wide knowledge of ophthalmology and its literature made him an ideal editor. So conscientious and painstaking was he that he read over everything and corrected mistakes. No amount of time was too great for him to spend, no detail too small to receive his personal attention; this in spite of

the fact that his greatest happiness was found in the home circle of his talented family—his wife, four daughters and son.

Axenfeld was regarded with great esteem, not only in his own country, but in other countries. He was a skilful and delicate operator, originated many refinements of technic, and was recognized as a wise consultant to whom many patients came for advice from all over the world.

At medical society meetings he was also a leader, not only on account of his scientific achievements, but also on account of his organizing ability and representative character. On Uhthoff's death in 1927, he was elected to be his successor as president of the Deutsche Ophthalmologische Gesellschaft, the outstanding German society. With Wessely, in 1927, he represented German ophthalmology in the conferences which reestablished the International Congresses of Ophthalmology, the first of which met at Amsterdam in 1929. At this meeting he was one of the foremost figures. It was a tired and older Axenfeld that we saw there, but his spirit was undimmed. He was still trying to find how he could do forty-eight hours' work in twenty-four. He spoke with sorrow of the fact that his multifarious duties seriously interfered with his loved research. It was a notable occasion in Amsterdam when the medal of the American Ophthalmological Society, established by his old friend Lucien Howe, was conferred on him. We shall not soon forget the delightful and eloquent response which he made in appreciation of the honor.

Throughout his life he kept in touch with his former students, mostly through postcards and letters of no mean length, almost always written in his own hand. Although he attended many meetings outside of Germany, it had never been possible for him to come to America, in spite of several warm invitations from our societies. In the winter and spring of 1930, in response to an invitation from the all-Japanese Medical Congress, he visited Japan, accompanied by his son, and gave several addresses on ophthalmologic subjects. This journey, on which he renewed many friendships, was a great joy to him, and it was hoped that it would afford the rest which he so sorely needed.

His death following an abdominal operation came as a great shock to his many friends and admirers, but his memory will long be cherished by those who follow him.

GEORGE S. DERBY.

# WALTER EYRE LAMBERT, M.D.
## 1859-1930

Dr. Walter Eyre Lambert died suddenly of heart disease on July 28, while enjoying his summer vacation at Murray Bay, Quebec. His health had not been robust for several months, following an attack of pneumonia in February, but his death came as a great shock to hosts of friends.

He was born in 1859 at Charleston, S. C., the son of Sarah Ann Murrell and of Walter Lambert, whose father was Walter Lambert of Castle Lambert, County Galway, Ireland. On his mother's side, Dr. Lambert was related to many old South Carolina families. Following a preliminary training at Charleston High School, where he graduated with honors, he obtained a scholarship in the College of Charleston, taking his B.A. degree in 1877.

His mother having died, his father returned to Ireland, as he had lost his property after the Civil War. Dr. Lambert followed him there in 1879. After four years at the College of Physicians and Surgeons in Ireland, the Carmichael College of Medicine and the Ledwich School of Medicine, he graduated from the Royal College of Physicians and Surgeons, Edinburgh. He began his study of the eye under Dr. Charles Fitzgerald and Mr. (afterwards Sir Henry) Swanzy.

In the summer of 1888, he went to Vienna and worked in the Fuchs clinic, returning to Dublin in the autumn to study with Swanzy. Early in 1889, he came to New York with an introduction from his late teacher to Dr. Richard Derby, who appointed him an assistant in his service at the New York Eye and Ear Infirmary. After the customary six months' probation, he was promoted to be assistant surgeon.

Dr. Lambert served the Infirmary for forty-two years, as attending surgeon (1896-1924), consulting surgeon (1924-1930), executive surgeon (1920-1922), director (1903-1930) and secretary to the board of directors (1903-1920). His devotion to this institution, his energy and his wise counsel were appreciated by his colleagues, who commemorated them in suitable engrossed and illuminated resolutions.

Elected a member of the American Ophthalmological Society in 1891, Dr. Lambert contributed to its proceedings from a wide clinical experience and engaged actively in its discussions. He was one of the first in this country to use and advocate the practice of skiascopy in the estimation of refractive errors, and he invented an instrument to simplify its application. He was chosen a member of the Council in 1920, and continued a member until 1924; he became vice-president in 1926 and president in 1927.

He was a Fellow of the American Medical Association, a member and former president of the New York Ophthalmological Society and the Hospital Graduates Club; twice chairman of the Section on Ophthalmology of the Academy of Medicine; a member and past president of the British Schools and Universities Club, a member of the University, Century and Calumet Clubs, and one time professor of ophthalmology in the New York Polyclinic Medical School. His early military instincts led him to become surgeon of the Twelfth Regiment, N. G., N. Y., in which he served for five years, leaving this corps to join the First Brigade as surgeon with the rank of Lieutenant-Colonel, and becoming a supernumerary after the Spanish-American War.

Possessed of great personal charm, Dr. Lambert enjoyed a large practice and was beloved by his patients; his Irish blood contributed a certain pugnacity to his nature—he never shrank from a fight—but he was quick to forgive, and sincere in his loyalty. Those who enjoyed his intimate acquaintance will miss its generous companionship, and deeply mourn a radiant soul.

JOHN R. SHANNON.

# Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

---

## Aqueous Humor

THE OCCURRENCE OF CITRIC ACID IN THE AQUEOUS HUMOR OF THE EYE. HERMAN GRÖNVALL, Acta ophth. **8:** 45, 1930.

By applying a method described by Thunberg for determining the presence of citric acid in fluids, Grönvall found that the aqueous humor of a rabbit's eye contained 50 micrograms of citric acid per cubic centimeter, or 0.005 per cent. The author could not demonstrate the presence of citric acid in the aqueous humor of the cow. He found it to be present in the eyes of human beings, hogs and calves.

A. M. YUDKIN.

## Bacteriology and Serology

THE BACTERIOLOGY OF THE CONJUNCTIVAL SAC IN DACRYOCYSTITIS BEFORE AND AFTER OPERATION. B. BAUMGART, Saggi di oftal. **5:** 567, 1930.

Smears and cultures from the conjunctival sac were made on serum agar the day before operation, from the sixth to the ninth day following operation and again after from twenty days to a month. In twenty-three cases simple excision of the sac was performed; in nine removal of the sac and lacrimal gland, and in seventeen dacryocystorhinostomy.

Pneumococci were present in from 56 to 33 per cent before operation, while a month afterward they were still to be found in about 15 per cent of cases. Streptococci persisted also in from 7 to 15 per cent of cases. The other organisms underwent marked reduction in number. The percentage of cases in which pneumococci persisted was especially high, 43, after combined excision of the sac and lacrimal gland. Although this was done in only seven cases, it may indicate the danger of deficient lacrimal secretion. On the basis of these studies, the author advises an interval of from ten to fifteen days between removal of the sac and any intra-ocular operation. The persistence of pneumococci and streptococci after dacryocystorhinostomy with a functioning opening into the nose is emphasized.

S. R. GIFFORD.

POSITIVE GONOCOCCAL COMPLEMENT-FIXATION REACTION IN SOME PATIENTS WITH OCULAR DISEASE. HOLGER EHLERS, Acta ophth. **8:** 64, 1930.

Ehlers found that a polyvalent antigen made of meningococci and gonococci proved beneficial in the diagnosis of ocular disturbances produced by the gonococcus. He reports in detail five cases in which the gonococcal complement-fixation test was helpful. It is not a method of treatment for gonorrhea.

A. M. YUDKIN.

## Conjunctiva

CONTRIBUTIONS TO THE STUDY OF VERNAL CONJUNCTIVITIS. L. MAMOLI, Saggi di oftal. **5:** 362, 1930.

To judge from the number of reports in the Italian literature, vernal conjunctivitis must be very common in Italy. Mamoli reports thirty-five cases seen during the year. These were examined with regard to constitution and signs of glandular imbalance. Many showed the combination of vasomotor lability, tachycardia and lymphoid hyperplasia which has come to be known as Angelucci's syndrome, and which is apparently identical with the syndrome of hypo-adrenia. The blood pressure was usually below 110; in some cases it was below 100. The urine in eight cases was examined for porphyrin, but was always found negative for this substance, which is evidence against the theory of photosensitization. Six patients were given skin tests for the various pollens, with negative results. The presence of vasomotor rhinitis was noted in twenty cases. The author was led to believe in the primary importance of constitution as the cause of vernal conjunctivitis, some combination of vagotonia, hypo-adrenia and hypogonadism being the only constant observations.

Treatment was by subcutaneous injections of epinephrine, combined with calcium intramuscularly and epinephrine-cocaine drops, the nose being observed for local symptoms.

S. R. GIFFORD.

LETHAL SEPTICEMIA DUE TO SEVERE STREPTOCOCCAL CONJUNCTIVITIS. E. LAZARESCU, Rev. med.-chir., Jassy **40:** 412, 1929.

A severe case of pseudomembranous conjunctivitis in a child, aged 2 years, is described. Streptococci and also scarce pneumococci were found in the secretion. The further clinical course showed ulceration of the cornea and panophthalmia refractory to any therapy. There was a rise in temperature to 39 C. (102.2 F.). The septicemia resulted in death at the end of a month.

N. BLATT.

## Congenital Anomalies

TWO CASES OF PERSISTENT PUPILLARY MEMBRANE. D. LAZARESCU, Rev. med.-chir., Jassy **40:** 136, 1929.

The pupillary membrane appears in the second month of intrauterine life and disappears in the fourth or fifth month. In rare cases—as it happened in both cases of the author—it may persist for life. In the ophthalmologic clinic of Jassy under direction of the professoress Dr. Puscariu, only two cases of persistent pupillary membrane were found among 22,000 patients. The first occurred in a boy, aged 14, the second in a student, aged 33, who gave a positive Wassermann reaction. Then follows the description of the various types which the pupillary membrane can present, among others adhesions to the lens, cornea or iris. Excellent slit-lamp illustrations demonstrate the close and instructive observations.

N. BLATT.

## Cornea and Sclera

HEREDITY IN KERATOCONUS. G. ABELSDORFF, Arch. f. Augenh. **103:** 293 (June) 1930.

The author traced the family histories of a few patients with keratoconus, and found that they had familial keratoconus. Two cousins of another patient were affected with the same disease. In one case both mother and daughter had keratoconus of both eyes. Another patient had an uncle and his two daughters who suffered from the same disease.

In none of the cases could the author find Fleisher's ring. All of his patients were young, between the ages of 15 and 25 years. The Zeiss contact lenses were tried by all of the patients, but only one could wear them for a long time.

The author gives an interesting point. All of his patients were living in the capitol of Colombia, Bagota, which is located 2,600 meters above the sea level, where keratoconus is a rather common disease.

O. SITCHEVSKA.

SUBSTRATUM OF THE RING OF DESCEMET'S MEMBRANE IN PSEUDOSCLEROSIS. J. KUBIK, Klin. Monatsbl. f. Augenh. **84:** 478 (April) 1930.

Two cases observed at Elschnig's clinic at Prague prompted the author to disagree with Vogt's opinion that the substratum of the pigment ring of Descemet's membrane in pseudosclerosis and Wilson's disease consists of silver. Neither does he accept the conclusion that the similarity of the color scheme is caused by one and the same chemical substratum. The solubility in cyanide of potassium, in his mind, is not convincing proof of the presence of silver, because gold and all the organic pigments are soluble in it. Kubik believes that in his two cases the substratum in question is organic and autogenous, i. e., deteriorated pigment (Abnutzungspigment) composed chiefly of lipoids. In more acute cases the pigment is deposited between the endothelium and Descemet's membrane, while in cases of long standing it moves in a mechanical manner into Descemet's membrane. This, in the author's opinion, is evidenced by the accumulation of the pigment in the portion of Descemet's membrane closest to the endothelium. Sunflower cataracts were not observed in either of Kubik's cases. (Abstracts of Vogt's articles can be found in ARCH. OPHTH. **2:** 480 [Oct.] 1929; **3:** 641 [May] 1930.)

K. L. STOLL.

RETURNS CONCERNING ONE HUNDRED AND SEVENTY-TWO CASES OF PARENCHYMATOUS KERATITIS. LAZARESCU-POPESCU, Rev. med.-chir., Jassy **40:** 41, 1929.

The author presents a statistical study referring to 172 cases of parenchymatous keratitis among 22,000 outpatients and 5,255 inpatients with diseases of the eye. Of the cases, 4.07 per cent were found to occur at from 1 to 5 years of age, 76.74 per cent from 6 to 20 years and 19.19 per cent from 21 to 55 years. As to the sex, 52.9 per cent of the patients were females. Heredosyphilis was present in 74.72 per

cent of the cases, and acquired syphilis in 8.14 per cent. Often parenchymatous keratitis is the unique manifestation of heredosyphilis. Hutchinson's triad was found in 3.4 per cent of the cases. Dystrophy of the teeth could be evidenced in 44.76 per cent, deformities of the bones in 30.23 per cent and a positive Wassermann reaction in 80.34 per cent of the cases.

N. BLATT.

LESIONS OF THE EYE DUE TO BLUE INK. NICOLAU, Soc. Român. de oftal., Bucuresti **10:** 554 (Feb.) 1929.

Nicolau reports the clinical observation of a case of strong injection of the eyeball, palpebral edema and severe pain. There was slight opacity of the cornea; the vision was 1/3. The discoloration of the cornea vanished after five days, but the epithelium of the cornea showed several exfoliations. At the end of fifteen days there was complete recovery. Instillations of the same ink into the eyes of rabbits provoked still severer lesions than they did in man. Later, the exfoliation of the cornea resulted in superficial necrosis with hypopyon.

N. BLATT.

## General

PANTHESIN, A NEW ANESTHETIC. R. PFIMLIN, Klin. Monatsbl. f. Augenh. **84:** 270 (Feb.) 1930.

Panthesin is an N-diethyl-leucinol-ester of the p-amino-benzoic acid, related to procaine hydrochloride. The author considers it especially useful in tonometry, when instilled in a 2 per cent solution, as it acts quicker than holocaine. It does not cause collapse, as cocaine may do, and is recommended for probing and irrigating the tear sac. A 2 per cent ointment is applied in chronic irritations, electric ophthalmia and erosions of the cornea, where it does not injure the stroma. It was used successfully in sclerotomies, iridodialyses and other operations on the globe, never causing hyperemia. Injections of a 0.5 per cent solution of panthesin with two drops of epinephrine to 0.5 cc. sufficed in the removal of chalazions, in xanthoma, in operations on the lid, extirpations of the tear sac and in tonotomies; doses of from 0.3 to 0.5 cc. were injected into the insertion of the muscle; from 5 to 8 cc. produced ganglion anesthesia for enucleation.

In the author's opinion, panthesin is a substitute for cocaine, and is void of toxicity or undesired actions and prevents secondary pain.

K. L. STOLL.

## General Diseases

CLINICAL RESEARCH ON THE RELATIONS BETWEEN OSMOSIS, BLOOD PRESSURE AND INTRA-OCULAR PRESSURE. F. POOS, Klin. Monatsbl. f. Augenh. **84:** 340 (March) 1930.

Undulations of the intra-ocular pressure have been found in juvenile diabetes, depending on the undulations of the percentage of sugar and water in the blood. The intra-ocular tension remained normal at from 20 to 21 mm. of mercury as long as the percentage of blood sugar was normal; tension exceeding 26 mm. of mercury is considered relative

hypertony and is caused by hypoglycemia. Undulations of the intra-ocular tension, due to changes of the osmotic blood pressure, are transient normally. In diabetic persons hyperglycemic and hypoglycemic changes are accompanied by disturbances of the percentage of water (decrease or retention), and by variations of the colloido-osmotic blood pressure. The increase of the relative contents of the serum albumin, causing an increase of the colloido-osmotic blood pressure, may be responsible for the permanent softening of the eye in diabetic coma, as described by Heine and Krause.

Parasympathetic irritations appeared in progressive hypoglycemia due to insulin, evidenced by miosis and spasm of the ciliary muscle; the irritations disappeared under feeding with carbohydrates. Changes of the volume of the retinal vessels in hyperglycemic precoma did not affect the function of the retina. Occasional local application of pilocarpine failed to influence the intra-ocular tension caused by osmosis. Undulations of the intra-ocular tension due to pathologic, spontaneous undulations of the concentration of the blood affect only glaucomatous eyes.

Injections of insulin may produce a sudden increase of the intra-ocular tension and must be watched, unless the pressure is normalized by an operation. Treatment with insulin can be undertaken without risk after a successful operation.

K. L. Stoll.

Fundus Changes in Leukemia. Sven Larsson, Acta ophth. **8**:22, 1930.

From a study of seven cases of myelogenous leukemia and an equal number of cases of lymphatic leukemia Larsson describes a composite picture known as retinitis leukemia. In the cases showing a choked disk lumbar puncture revealed increased intracranial pressure. The author attributes the fundus changes, choked disk, edema and hemorrhages to the pressure of the fluid in the cranial cavity on the optic nerve and vascular system. The congestive phenomenon in the fundus is frequently accompanied by signs of renal deficiency. The significance of renal lesions and increased intracranial pressure in the etiology of fundus changes in leukemia is still doubtful.

A. M. Yudkin.

## General Pathology

A Note on the Hydrogen Ion Concentration of the Vitreous in Eyes Affected by Naphthalene. D. R. Adams and P. T. Kerridge, Brit. J. Ophth. **14**: 397 (Aug.) 1930.

It has been shown (Adams, 1930) that naphthalene cataract is almost invariably preceded by the formation of excessive retinal exudates, an edematous change that might easily affect the vitreous in such a way as to alter its hydrogen ion concentration. It was therefore considered worth while to investigate the hydrogen ion concentration of the vitreous in rabbits at different times after the administration of naphthalene, in order to see whether it exhibited any change in hydrogen ion concentration which might account for the local condition of the lens or could be correlated with the edematous changes in the retina. At the same time it was interesting to find the hydrogen ion concentration of the normal vitreous, since it has not been determined

previously for the rabbit. The conclusion reached was: The hydrogen ion concentration of the vitreous does not appear to change at any moment after the administration of naphthalene in such a way as to account for the development of cataract; neither is it affected by other intra-ocular changes, such as retinal edema or atrophy.

W. ZENTMAYER.

PARTIAL EPITHELIAL LINING OF THE ANTERIOR CHAMBER AFTER CATARACT OPERATION. F. PAPOLCZY, Klin. Monatsbl. f. Augenh. **84:** 266 (Feb.) 1930.

A woman, aged 68, had a catract removed in 1911, and a fine secondary membrane split in 1916. She returned in 1927, stating that the eye had been inflamed for four years and that vision had failed gradually. The soft atrophic and amaurotic globe was removed. Pathologic examination showed the entire surface of the iris and the pupillary area covered by several layers of plate epithelium originating from Descemet's membrane near the corneal scar. Proliferation of the epithelium into the scar could not be discovered.

In the author's opinion, the formation described, which is related to cysts of the iris, is not caused by chronic iridocyclitis, but is due entirely to filtration of the vitreous humor through a slowly closing operative wound.

K. L. STOLL.

## Glaucoma

THE FUTURE OF IRIS-INCLUSION IN GLAUCOMA. H. HERBERT, Brit. J. Ophth. **14:** 433 (Sept.) 1930.

In this paper is given the basis of a firm faith, strengthened by recent experience, in iris-inclusion as inevitably the future treatment for many chronic primary glaucomas, and a new and much needed item in operative technic that alters the outlook materially.

In the author's experience, late results of trephining done by various surgeons suggest that in eyes thus operated on, in which there is no conjunctival vesiculation, there is a greater liability to recurrence of tension than in eyes with linear filtering scars secured by either iridectomy or small flap sclerotomy. Herbert has hesitated to recommend expressly iris-inclusion because of failure to get rid entirely of the conjunctival flap with its presumptive risk of late infection, and because of the need for insistence on measures to be taken to eliminate the risk of sympathetic ophthalmitis.

It can at last be claimed that the needful control over conjunctival changes has been secured, from observations of the effects of rare instances of unintentional iridodialysis produced during operations for glaucoma. Iridodialysis is effective in securing localized fibrosis in the conjunctiva covering an iris prolapse, extending down into and practically replacing the iris tissue, yet not interfering in the least with effective drainage of aqueous into surrounding subconjunctival tissue. Elimination of sympathetic ophthalmitis can be brought about in operations for glaucoma only by the use of sufficiently strong yet harmless antiseptic preventive measures. It must be remembered that this protection, afforded by antiseptic applications before operation, is definitely time limited.

The use of minor precautions, such as expression of meibomian secretions, swabbing the margin of the lid, the use of a type of lid speculum that covers the lashes, placing the conjunctival puncture from 6 to 7 mm. from the corneal margin, rapid rinsing of the tips of the iris forceps or scissors in boiling water should they have to be introduced into the wound a second time, is recommended.

Careful details are given as to the preoperative treatment and the method of using antiseptic solutions and anesthetics at the time of the operation. The site of the incision is often to one or the other side, either to avoid the larger blood vessel or possibly because the patient does not look sufficiently downward. The edge of a small blade keratome pushes the conjunctiva from a point 6 to 7 mm. from the corneal margin, down nearly to the corneal margin, the exact point depending on the varying degree of overlapping of the cornea by the conjunctival limbus. The point of the knife is pushed on into the anterior chamber, at first inclined toward the iris, very slowly and preferably with side to side movements, until an incision of desired size is produced. The ordinary pattern of blunt iris forceps is introduced through the subconjunctival pathway; the iris is well gripped and pulled slowly toward the center of the pupil until is it seen to have been torn from its base locally. It is then drawn up through the corneoscleral wound, to be cut radially under the conjunctiva, preferably with straight iris scissors. Repeated snips are made because the effect cannot be seen sufficiently, often being hidden by blood and iris pigment under the conjunctiva. It is not essential that the cut should reach the pupillary border, at which one aims. The iris is not released until the cutting is complete. In acute glaucoma the author favors the subconjunctival iridectomy that can be made, leaving the iris incarcerated.

W. ZENTMAYER.

THE RETENTION OF INTRA-OCULAR PRESSURE FOLLOWING PUNCTURE OF THE ANTERIOR CHAMBER IN GLAUCOMA. PETER C. KRONFELD, Ztschr. f. Augenh. **71:** 48 (April) 1930.

Kronfeld shows that in glaucoma simplex with increased pressure the restoration of the intra-ocular pressure is apparently more rapid than in normal eyes. When the original pressure is attained, it is followed by a phase of reactive increased tension, which is finally followed by a transient and insignificant reactive decrease in pressure. Glaucoma simplex (degeneratio glaucoma optici) (Elschnig) behaves like glaucoma simplex with increased pressure. The rapid restoration of original pressure after puncture can be considered as pathognomonic of glaucoma simplex. The rapidity with which intra-ocular pressure increased from 0 to 15 mm. of mercury may be considered as a measure for the sum of causative factors which take part in the restoration of the intra-ocular pressure, i. e., vasomotor reaction, the alteration of the production of the aqueous humor and the abnormal protein content of the fluid. As the acceleration of the pressure is increased in glaucoma simplex, the so-called causative factors are increased and hence must be a part of the glaucomatous hypertension. Kronfeld confirms the assumption of others, that hypdrophthalmos is a retention glaucoma and secondary glaucoma is a process of retention plus inflammatory hyperemia of the uveal tract.

A. M. YUDKIN.

## Injuries

NOTES ON B. WELCHII INFECTION OF THE GLOBE. J. B. HAMILTON, Brit. J. Ophth. **14:** 452 (Sept.) 1930.

Two further cases of infection with *B. welchii* are recorded. Both occurred in men and resulted from the entrance of a foreign body into the eye. It was seen in the vitreous with a large air bubble attached.

In the first case the foreign body was removed eleven hours after the accident. Twenty-four hours later evisceration was done because of panophthalmitis with constitutional symptoms. In the second case, the foreign body was removed eighteen hours after its entrance, and antigas gangrene serum was administered; four hours later the eye was eviscerated, panophthalmitis with general symptoms having developed.

The author states that from his experience *B. welchii* infections of the eye may be divided into two groups: (1) a mixed infection of *B. welchii* and some pus-forming organism, to which the first case belongs and (2) pure infection of *B. welchii,* to which Heath's and his own second case belong. Briefly, the general appearance of the first group is that of an ordinary panophthalmitis, while the general symptoms of the second group are those of an abdominal crisis.

The prognosis is remarkably good in both groups. This is in contradistinction to the injury received during the world war, in which the outlook was so grave and death so rapid. Perhaps the favorable prognosis is due to the perfect excision of infected tissue consequent on simple evisceration. Serum should nevertheless be administered.

W. ZENTMAYER.

INJURIES OF EYE OCCURRING IN SKIING: THEIR PREVENTION. J. STREBEL, Schweiz. med. Wchnschr. **60:** 391 (April 26) 1930.

Strebel asserts that crushing and laceration of the tendons of the muscles of the eye are the most frequent injuries in skiing. Tearing of the tendon of the inferior oblique muscle often results in a permanent paralysis and narrows the field of vision. A crushing of the lateral rectus muscle with extravasation of blood under the conjunctiva and commotio retinae leads to temporary paralysis and other complications. However, they often disappear again spontaneously. Conditions are considerably more complicated when the bony walls are fractured and when the periosteum and Tenon's capsule are lacerated. Injuries in the region of the nasal canthus often result in a tearing of the lacrimal canal with subsequent lacrimation and chronic conjunctivitis. Occasionally the inferior oblique muscle is torn off, and sometimes the accessory nasal cavities are opened. The latter injury may lead to sinusitis, and to meningitis. If the blow from the ski stick is very severe, the optic nerve, the ciliary vessels and the medial rectus muscle may be lacerated and the entire eyeball is torn out. Most injuries of the eyes occurring in skiing are caused by the ski sticks. In order to avoid these traumas, the author emphasizes that only padded sticks should be used. He also stresses that the ski sticks should not be too long. Their length should not exceed that of an ordinary walking stick. Beginners should learn to ski without the aid of sticks.

[J. A. M. A.]

## Instruments

TONOMETRY AND THE VARIOUS INSTRUMENTS EMPLOYED IN ITS PRACTICE. R. I. LLOYD, Am. J. Ophth. **13:** 396 (May) 1930.

The different methods and instruments for taking the tension are described, and the history of tonometry is reviewed. Illustrations of the older and obsolete instruments, and also the more recent and complicated instruments, are shown. The methods of using these and the principles involved are discussed.

W. S. REESE.

## Lens

A PECULIAR FORM OF CONGENITAL CATARACT. N. COLLEVATI, Saggi di oftal. **5:** 391, 1930.

The author saw this condition in a mother and two sons. The opacity was almost identical in the three cases, and consisted of a series of spicules extending from the anterior embryonal sutures, with their peripheral ends broadening into a truncated conical shape. Vision varied from 8/10 to 3/10, the difference depending on slight variations in the size of the opacity. The mother showed in addition some opacities in the anterior cortex of the coronary type. Since the central opacities were confined to the embryonal nucleus, they must be considered to have formed some time before birth. Careful examination of the blood chemistry disclosed a hypocholesterinemia in all cases, which suggests to the author a change in lipoid metabolism possibly due to some disorder of the liver.

S. R. GIFFORD.

SENILE CATARACT, ARTERIAL HYPERTENSION AND EXPULSIVE HEMORRHAGE. PUSCARIU, Soc. Român. de oftal., Bucuresti **10:** 552 (Feb.) 1929.

Among 989 extractions of cataract done in the Clinic at Jassy, there were eight cases of expulsive hemorrhage. Two of the patients had hypertension. In 23.9 per cent of all of the cases of cataract there was evidence of hypertension in the arteries, but expulsive hemorrhage did not occur in any of them. Systemic hypertension cannot be considered as the cause of expulsive hemorrhage, nor can the latter be avoided if an attempt is made to lower the hypertension before operation.

N. BLATT.

## Methods of Examination

THE VALUE OF THE INTRACUTANEOUS TUBERCULIN TEST OF MANTOUX FOR THE DIAGNOSIS OF TUBERCULOUS DISEASES OF THE EYE. R. BRAUN, Arch. f. Ophth. **124:** 182 (April) 1930.

This article represents the answer of the University eye clinic in Berlin to the recent publication of von Frisch and Pillat, who considered the Mantoux test a valuable diagnostic means for the detection of tuberculous diseases of the eye. Braun did the Mantoux test on 200 patients, some with definite tuberculous lesions, others with lesions of unknown etiology and still others with lesions that could with great certainty be considered nontuberculous. The result was that, among

the patients with definite tuberculous lesions, great sensitiveness to tuberculin was about 20 per cent more frequent than among those who had definite nontuberculous lesions. In the latter group, however, were a great many—30 per cent—who gave a positive reaction to 0.1 cc. of a 1:1,000,000 dilution of tuberculin. Therefore, Braun concludes that a positive Mantoux test does not justify the diagnosis of tuberculous disease of the eye, even if all the other etiologic possibilities can be ruled out.

P. C. KRONFELD.

## Ocular Muscles

A NEW "EYE AND HAND" METHOD OF TRAINING SQUINTS. E. E. MADDOX, Am. J. Ophth. **13:** 379 (May) 1930.

The author presents a new instrument, the cheiroscope, designed to have patients with squint employ their hands to educate the squinting eye, imitating the natural process of infancy in which the hand and eye mutually perfect their training by trial and error. The new instrument is similar to a stereocampimeter. The author replaces the carrier of a stereoscope with a horizontal surface on which the child can draw or play, using the hand to demonstrate or develop the act of fusion of the image or object controlled by the amblyopic eye with that of the normal eye. A mirror arrangement is used to obtain the adjustment necessary in the case of convergent squint. The author gives a number of devices used to develop the fusion faculty. He gives a number of general considerations necessary for training in cases of squint.

W. S. REESE.

THE SURGICAL TREATMENT OF CONCOMITANT STRABISMUS. F. SCARDAPANE, Saggi di oftal. **5:** 164, 1930.

The author reports the cases of twenty-four patients operated on in the di Marzio Clinic. Resection of the muscle was practiced in all cases on one or both eyes, aided by tenotomy only in severe cases. Success was obtained in all cases of divergent squint, 57 per cent of convergent squint and 77.5 per cent of alternating squint. In the others a slight deviation remained, exceeding 20 degrees in only one case. When tenotomy was not performed, bilateral resection was usually necessary, and this procedure, the author believes, provides the best results. Orthoptic exercises were used after operation, and in a number of cases vision in the emblyopic eye improved notably, from 1/60 to 1/10 in one case, from 2/10 to 5/10 in another case and in appreciable amounts in two other cases.

S. R. GIFFORD.

## Orbit, Eyeball and Accessory Sinuses

DESTRUCTIVE PURULENT OPHTHALMIA ACCOMPANYING AN ERUPTIVE FEVER WITH STOMATITIS. J. M. WHEELER, Am. J. Ophth. **13:** 508 (June) 1930.

In a Jewish boy, aged 8 years, after he was caught in the rain, generalized aches and pains developed. Two days later his eyes became

infected, his nose and mouth were dreadfully inflamed and a rash broke out. A diagnosis of erythema, possibly of drug origin, was first made. Dr. A. M. Stevens diagnosed the condition as one described by him and F. C. Johnson in 1922, under the title "A new eruptive fever associated with stomatitis and ophthalmia." The patient's eyelids became swollen shut, with pus streaming from them. The nares were excoriated, and adherent to the lips were heavy, dark crusts and fresh blood. Brownish spots were scattered over the body. The temperature ranged from 98 to 101 F. and the pulse rate from 80 to 120. Urinalysis, examination of the blood and the Wassermann test gave negative results. Both corneas became ulcerated so that the iris could not be seen. Culture of a smear from the eye showed short chain, nonhemolytic streptococci and gram-negative diplococci, probably not gonococci. Both corneas perforated, and sight was gone in one eye and almost so in the other. The author discusses the possible diagnosis and suggests a distinct disease not hitherto recognized. W. S. REESE.

BRAIN ABSCESS AND MENINGITIS PRODUCED BY AN ORBITAL ABSCESS. LAZARESCU-VARLIC, Soc. Român. de oftal., Bucuresti **10:** 554 (Feb.) 1929.

Lazarescu-Varlic reports a case of injury resulting in an orbital phlegmon which produced meningitis and caused a fatal issue after eighteen days. Autopsy revealed, besides suppurative meningitis, an abscess of the brain in the left frontal lobe. N. BLATT.

## The Pupil

PUPILLARY REACTIONS OF PREGNANT AND NONPREGNANT WOMEN AND PRACTICAL APPLICATION TO DIAGNOSIS OF PREGNANCY AS SIMPLE PROCEDURE. Z. BERCOVITZ, Am. J. Obst. & Gynec. **19:** 767 (June) 1930.

Series of pregnant and nonpregnant women have been studied by Bercovitz to note the pupillary reactions of each group following the instillation of their own serum into the conjunctival sac. In seventy-two nonpregnant normally menstruating women, taken at all stages of the menstrual cycle, it was noted that there were no changes in the pupil as a result of instilling the patient's own serum into the conjunctival sac. In twenty-six nonpregnant women who had not menstruated for any reason whatever, whether endocrine disturbances, menopause or after pelvic operation, only two gave positive reactions to their own serum. Thus, in a total of ninety-eight patients, only two gave positive reactions; the others were negative. Of sixty-eight pregnant women it was found that in 80 per cent of the observations there was a change in the size of the pupil. This change was a dilatation, a contraction or, as in a few cases, only a marked activity of the pupil. None of these changes occurred in nonpregnant women. The pupillary reactions to epinephrine hydrochloride were studied in fifty-four pregnant women. In these it was found that 75.9 per cent reacted with changes in the size of the pupil. Of ninety-eight nonpregnant women only two cases reacted positively when epinephrine hydrochloride was instilled into

the conjunctival sac. Nineteen women who gave positive pupillary reactions ante partum were also studied post partum. The postpartum observations were made with serum and in each case the reaction was negative. Twenty observations were made with epinephrine; of these, fourteen were entirely negative, two questionable and four positive. The pupillary reactions of forty-six questionable cases have been studied. Of these, subsequent physical examinations revealed the fact that twenty were pregnant and twenty-six nonpregnant. Of the pregnant women, 62 per cent reacted positively to their own serum. In most of these cases it was impossible to make a diagnosis by physical examination at the first visit of the patient and the diagnosis was confirmed by subsequent physical examinations.

[J. A. M. A.]

## Physiologic Optics

THE INFLUENCE OF THE LIDS ON THE CURVATURE OF THE ANTERIOR SURFACE OF THE CORNEA. FREDRIK BERG, Acta ophth. **8:** 1, 1930.

By measuring the surface of the cornea with a modified Javal-Schiötz ophthalmometer, Berg noted that the central portion of the cornea is almost globular, that the greatest sloping is upward and that there is a definite difference between the vertical and horizontal meridians. The former plane in normal eyes is quite regular in its curvature, whereas in the latter it varies. The upper part of the vertical plane may show an abrupt sloping while the lower portion is more constant. By skiascopy a line described by Handmann is frequently discerned. This aberration is probably due to the pressure of the upper lid on the surface of the cornea. He also noted that in patients with chronic conjunctivitis and blepharitis, the curvature is altered considerably. Frequently patients presenting the so-called palpebral line show a marked wrinkling of the forehead. Some Japanese observers describe monocular diplopia in these cases. Bull, in 1894, reported a similar line in the pupillary area and described a method for studying this phenomenon. With increasing age it was noted that a corneal astigmatism often decreased or was in inverse proportion to the original condition. Gullstrand attributed this change to increasing rigidity of the globe and a diminution of palpebral tonus. The Handmann line was rarely observed in senile persons.

A. M. YUDKIN.

## Physiology

THE INFLUENCE OF THE VISUAL CORTEX ON VISUAL ACUITY. S. W. KRAVKOV, Arch. f. Ophth. **124:** 76 (April) 1930.

If black targets on a white ground are used for the determination of monocular visual acuity, the latter can be improved considerably if light falls into the other eye. If the targets are white on a black ground, the visual acuity is diminished by light falling in the other eye. These influences on the visual acuity of one eye during stimulation of the other eye last several minutes, during which the visual acuity gradually returns to the level of strictly monocular vision. If the point in the eye of which the acuity of vision is being tested does not correspond to

the point in the other eye, which is just stimulated by light, the acuity of vision is not altered by the process in the other eye. These experiments show that the threshold for the perception of two objects as separate objects depends to a certain extent on the state of activity of the visual cortex.

P. C. KRONFELD.

FURTHER OBSERVATIONS ON AMMONIA FORMATION IN THE RETINA. HANS RÖSCH, Ztschr. f. physiol. Chem. **186:** 237, 1930.

Further experiments on the influence of light on the amount of ammonia formed in the retina showed that in early spring and summer there was far less difference between the dark adapted and the illuminated eye than previously. This is apparently due to the greater amount of light which was falling on the retinas at this time of year, leading to a considerable increase in the formation of ammonia.

Simultaneous illumination of both isolated eyes of a frog leads to a similar marked increase in the ammonia content. The amount of illumination necessary to produce an increase in the formation of ammonia could be reduced in time and in intensity; an exposure of about two minutes was found essential to produce an effect. Experiments were undertaken, different portions of the spectrum being used, and it was shown that the ultraviolet rays of around the region of 365 were ineffective.

F. H. ADLER.

CIRCULATION OF BLOOD IN THE RETINA. P. VANCEA, Cluj. med. **11:** 117 (March) 1930.

After discussing the literature on investigations and observations on circulation of the blood in the retina and blood pressure in the retinal arteries, the author describes his own researches in which he used the method of Baillart. He applied Baillart's dynamometer to the temporal portion of the eyeball, and at the same time observed the retinal vessels ophthalmoscopically. The pressure on the globe was then increased by means of the apparatus until the arterial pulsation became perceptible (minimal or diastolic compression); then the pressure was increased until pulsation ceased (maximal or systolic compression). Of these two compressions only the first, the diastolic one, is constant and dependable. These ophthalmologic investigations conducted in widely different internal, neurologic and gynecologic systemic diseases, permitted him to observe hypertension and hypotension in the retinal arteries. Hypertension was found to be concomitant with diseases of the heart, aorta and kidneys, but also to occur independently of any renal or vascular disease. When hypertension was associated with the aforesaid diseases, optic neuritis and neuroretinitis with hemorrhages were frequently found at the same time. In some cases, however, other lesions were absent, the retinal arterial hypertension constituting the only ocular symptom. The subjective symptoms of this hypertension varied greatly but were generally the same as those complained of in diseases of the optic nerve. The evolution of retinal hypertension is followed, as a rule, by the evolution of the concomitant systemic disease. In pure retinal hypertension no other changes of the eye-

grounds are met with; these eyes are also normal as regards function. In most of the cases retinal arterial hypertension is a manifestation of coexisting intracranial hypertension or of general arterial hypertension.

He also found arterial retinal hypotension as frequently as arterial retinal hypertension. This hypotension is found almost constantly in normal pregnancy and in pulmonary tuberculosis—in the first case, independent of systemic circulation, and in the second case, governed by the general arterial tension. In endocardial, myocardial and pericardial diseases there was likewise retinal hypotension in connection with the systemic circulation. Along with retinal hypotension there was sometimes in the same eye optic neuritis with edema, retinal phlebitis and atrophy of the optic nerve. Pure cases of retinal hypotension are supposed to be responsible occasionally for idiopathic stubborn headache.

N. BLATT.

## Retina and Optic Nerve

ON LOOKING FOR HOLES IN DETACHED RETINA. J. B. HAMILTON, Brit. J. Ophth. **14:** 455 (Sept.) 1930.

This is a practical paper of much interest. The author divides cases of detachment into two groups: 1. Those in which he does not expect to find a hole; (*a*) sarcoma of the choroid (several patients with such cases have been sent half way across Europe for ignipuncture, only to be returned with a polite note), and (*b*) shallow inflammatory detachments. 2. Those in which he expects to find a hole. This group includes myopic and the traumatic detachments, or a combination of both. A careful history is often of value in the finding of holes. The subjective localization of a scotoma in the field may indicate where to look for the hole. Dilatation of the pupil is essential, as most holes are anterior to the equator. The author prefers the direct method of ophthalmoscopy. There are three main types of holes: (1) tears at the ora serrata, which are often detected only by the contrast of their white folded posterior edge and the lesser white of the main detachment; (2) small round holes, which appear as if they had been punched out; these are often associated with old choroiditis and are usually to be found about from 1 to 1.5 disk diameters from the ora serrata, and (3) horseshoe rents, which are usually found in cases with a traumatic history and vary tremendously in size.

If one finds an old patch of choroiditis, its vicinity should be diligently scrutinized and possibly much time will be saved.

Multiple holes are by no means uncommon and must be searched for. Fresh holes may appear after ignipuncture. Secondary holes usually appear as rents at the ora serrata. Invariably, if the detachment is above the retina, the hole is there also. It may be covered by a fold, and uncovered by putting the patient to bed, for three or four days. If the detachment is localized to a medial or lateral position, the hole is usually on that side in the upper third of the detachment. As most detachments usually settle to this position, it is difficult to say when seen for the first time and below, where the hole will be found.

If at first one fails to detect a hole, the patient should be put to bed. A change in the position of the body for a short time often means a change in the position of the detachment.

Patches of atrophy in the detached retina may be mistaken for holes. The author thinks that fluid from the vitreous can penetrate through them, and that they are therefore potential holes. A cleft below often divides the detachment into symmetrical portions. As the cleft looks red and the billowy retina on each side looks white, it may be mistaken for a hole. In aphakia, the detachments usually occur from 30 to 40 years of age, after needling for lamella cataract. Finding holes in these is a superhuman feat, but they have been seen.

W. ZENTMAYER.

AMAUROTIC FAMILY IDIOCY AMONG THE JEWISH CHILDREN OF WHITE RUSSIA. S. LEWIN, Arch. de oft. de Buenos Aires **5:** 73 (Feb.) 1930.

This paper is based on the study of four cases from the pediatric clinic of Professor Lecnow and from the laboratory of Professor Krol of Minsk. In three cases autopsy was performed and histologic examination made. This series was supplemented by ten other cases supplied by Drs. Epstein, Kliwanskaja, Bruck and Monjukowa. After a thorough and painstaking study from clinical and anatomic points of view, the author arrived at the following conclusions: 1. Although the etiology of amaurotic family idiocy is still not clear, it is to be noted that the majority of cases occur in the same family and among Jews, thus confirming a hereditary family predisposition. 2. It is a disease of the nervous system brought about by congenital weakness, to which is added an incapacity of the nerve cells to carry out their normal metabolism. 3. The hereditary character of the disease is recessive. 4. This disease cannot be considered as a sign of atavism. 5. From a eugenic point of view, if consanguineous marriage favors a disappearance of the disease by the death of the affected offspring, it must not be forgotten that it can be transmitted by apparently healthy offspring. 6. The histologic lesions found were swelling of the cellular bodies with globular broadening of the nerve fibers, and increase of the glia without any mesodermal reaction. It is yet to be determined whether these lesions are due to fermentative processes or to other endogenous lesions.

C. E. FINLAY.

RECENT EXPERIENCES WITH IGNIPUNCTURE IN DETACHMENT OF THE RETINA. A. VOGT, Klin. Monatsbl. f. Augenh. **84:** 305 (March) 1930.

A detailed report on seventeen new cases is preceded by the continuation of the histories of the eight cases published in the *Klinische Monatsblätter für Augenheilkunde* (**82:** 620 [May] 1929; abstr., *Arch. Ophth.* **2:** 599 [Nov.] 1929). In the first series a fifth cure was effected, one additional improvement was obtained and two further cases had to undergo a second ignipuncture. Three cases remained cured for two and one-half years and longer. The eighth case, that of a woman, aged 58, with a myopia of 11 diopters, is remarkable: The first ignipuncture was followed by complete detachment of the retina, and four more attempts, which failed, did not prevent the sixth ignipuncture to be so successful that the patient has a vision of 0.2 and can read newspaper print at close range.

Pathologic changes of the vitreous and its structure, found almost uniformly in every case, are the probable causes of the formation of holes and ruptures in the retina.

The first group of the new series comprises six especially grave cases in which no satisfactory results could be obtained owing to the size of the holes, the different complications, the ages of the patients or their refusal of a second operation. Some features of interest met with in this group may be pointed out: In one case, that of a woman, aged 46, in which a spot, only suspected to be a rupture, was cauterized, reattachment of the retina was obtained. Vision still remained 5/15 when the patient was examined twenty months later. In another case, that of a woman with a myopia of 15 diopters, multiple ruptures were localized and cauterized by two ignipunctures in one sitting. The closure of one of two holes by ignipuncture was sufficient to bring about a cure in one case, while in another six ignipunctures were performed in a myope of 15 diopters with eleven holes in the retina, with the result that the vision was improved to 5/20 after the operation and had increased to 5/15 three months later. In the case of a woman, aged 31, who had been kept on her back for eight months, after a detachment had developed in the second eye, there were found nine holes which were closed by two extensive cauterizations performed at the same sitting The vision was normal two weeks after the operation and remained perfect four and one-half months later. No less remarkable is the case in which improvement was obtained after cauterization of a rupture 10 mm. long and 4 mm. wide. Four plates and about thirty-six colored pictures illustrate the shape and size of the holes and ruptures in the retina.

An important part of the publication consists of chapters on the genesis of spontaneous detachment of the retina and on localization and diagnosis of holes and ruptures of the retina, including detailed calculations for this purpose. Special consideration is given the technic of the operation and its combination with temporary resection of the external muscles of the eye. Descriptions of the preparation for the operation and of the course of the after-care furnish valuable hints.

A new feature is the retrobulbar ignipuncture. It is performed by means of an especially long, thin wire loupe to be introduced through the sclera without previous use of a knife. This wire is guided along the inside of another new instrument, Vogt's so-called "protecting spoon." These two instruments render ignipuncture possible at close proximity to the disk and the macula.

K. L. Stoll.

## Tumors

A Case of Epibulbar Basal Cell Carcinoma with Penetration of the Globe. B. W. Key, Am. J. Ophth. **13**: 187 (March) 1930.

A man, aged 62, complained of a rapidly growing "sore" at the inner angle of the right eye which was first noticed six months previously as a small, ulcer-like thickening at the junction of the conjunctiva of the caruncle and the adjoining skin. Examination showed a large, ugly, fleshlike ulcer with highly vascularized and thickened margins, occupying the nasal portion of the right orbit and conspicuously sym-

metrical. It appeared as a three-petal, leaflike formation encroaching on the nose. In the canthus there was loss of substance, extending to the ciliary region of the globe. The cornea showed a comparatively rapid edema and basal cell invasion of the corneal stroma, beginning at the nasal limbus and, at the same time, occupying the nasal iris angle and progressing temporally as a whitish cloud in the corneal substance. When this affected the patient's vision, he finally consented to operation. Coincident with this rapid advance of the globe, exophthalmos and exotropia occurred. The time consumed in the advance of the growth from the iris angle into the iris and across the cornea to the pupillary center was about fifteen days. The orbit was exenterated. Three months after his discharge the patient returned complaining of an enlargement of the glands of the right side of the neck. The orbit showed no evidence of recurrence. Eight photographs show the growth and the manner in which it invaded the ocular structures. The author discusses carcinoma and makes the following summary, the last point of which he stresses particularly. 1. The primary location of the carcinoma was in the angle of the nasal commissure. 2. Its exceedingly rapid advancement was similar to that of an infection, changes being noted daily, especially in the layers of the deep cornea. 3. Its pathway of penetration of the wall of the globe along the anterior ciliary vessels, apparently without involving the deeper nonresisting orbital structures, is noted. Penetration occurred with a curiously destructive influence on the two chiefly resisting membranes of the globe—both sclera and Descemet's membrane. Its penetration without vitreous changes, until the last few days before exenteration, is an interesting observation as evidence of the resistance of the choroid to carcinomatous invasion. 4. Its symmetry of development as it invaded each structure in its path—lids, conjunctiva, sclera and cornea—may be attributed to its primary location followed by the mechanical irritation of the lids and by the act of convergence of the globe.

W. S. Reese.

Melanoma of the Choroid, Coexisting with Tumor on Chest Wall. G. Freiman, Am. J. Ophth. **13**: 521 (June) 1930.

A man, aged 64, who was unable to see for one month, showed a retinal detachment in the left eye in the inferior temporal quadrant extending well down to the periphery. A smaller bulge nasally hid the disk. Transillumination gave negative results. The Schiötz tonometric reading was 20 mm. for the right eye and 12 mm. for the left. Two and a half weeks later a scleral trephining was done in the lower temporal quadrant. Ophthalmoscopic examination while the patient was still on the operating table showed that the detachment had only partially collapsed, and a well defined mass presented in the center of the field. Twenty-four hours later this mass appeared as a large domelike elevation coming abruptly well forward, clearly defined and a dirty yellow, with many vessels coursing over its surface and giving the impression of solidity. Physical examination disclosed a tumor on the anterior wall of the right side of the chest between the fifth rib and the costal margin, which was thought to be a malignant sarcoma. The eye was enucleated and found to contain a melanoma apparently arising from the choroid.

W. S. Reese.

# Society Transactions

EDITED BY DR. GEORGE S. DERBY

## AMERICAN OPHTHALMOLOGICAL SOCIETY

*Sixty-Sixth Meeting, Hot Springs, June 24, 1930*

EMORY HILL, M.D. *Secretary*

WILLIAM CAMPBELL POSEY, M.D. *Chairman*

TRANSPLANTATION OF THE HUMAN CORNEA. DR. BEN WITT KEY, New York.

Two patients who were clinically suitable for the attempt at corneal transplantation and who presented themselves at the New York Eye and Ear Infirmary afforded the opportunity for this rare experiment in operative technic.

The procedure in preparation and in operation was described. Complications arising after operation were discussed. The factors believed to have contributed to the complete take of the transplant were presented. Three illustrations were shown.

DISCUSSION

DR. H. W. WOOTTON, New York: Although the operation has often been performed, the visual results have been universally poor. The best result appears to have been in von Hippel's case, in which there was vision of 20/200 one year later.

DR. W. E. LAMBERT, New York: I believe that it is almost futile to attempt these operations on account of the poor visual results.

DR. JOHN W. BURKE, Washington, D. C.: Dr. Burke spoke of the technic and of a patient who retained enough vision to walk about for six years. The eye was subsequently lost from secondary glaucoma.

DR. W. R. PARKER, Detroit: In England recently I saw J. W. Tudor-Thomas perform some transplantations in rabbits. Trephining was employed, and it was found expedient to cut the disks a little smaller than the areas they were to be placed in. There were four complete successes in many operations.

DR. W. B. DOHERTY, New York: Ebeling and Carrel, in 1921, performed a successful corneal transplantation from one cat to another.

TRIBROMETHANOL (AVERTIN) AS AN ANESTHETIC IN OPHTHALMIC SURGERY. DR. WILLIAM H. WILMER, Baltimore.

Dr. Wilmer described the use of tribromethanol as an anesthetic in ophthalmic surgery as follows: the making of the solution, the preparation of the patient, the dosage, the administration, the contraindications, the advantages and the disadvantages.

DISCUSSION

DR. W. R. PARKER: I used tribromethanol in three cases. The results were satisfactory in two cases; in the third, ether had to be added, but the amount was considerably less than would otherwise have been necessary.

DR. H. G. PRESTON, Harrisburg, Va.: The following figure for tribromethanol anesthesia were supplied by the company that makes the drug: 35 deaths in 300,000 cases; 24 of these were regarded as preventable, and 11 as unpreventable. I endorse tribromethanol.

DR. T. B. HOLLOWAY, Philadelphia: How deep is the narcosis? Can the depth be estimated and controlled?

DR. FRED A. DAVIS, Madison, Wis.: Tribromethanol anesthesia was used in twenty-nine operations on the eye. Most of the patients had received previous medication, such as morphine or atropine. Seven of the twenty-nine required supplementary anesthesia, usually nitrous oxide, but two required ether. All received cocaine 4 per cent locally, introduced into the cul de sac. The time of sleeping after the patient returned to the wards varied from three to fifteen hours. Nine patients required postoperative medication, usually codeine and acetylsalicylic acid. Five patients were very restless, four requiring restraint by straps; twenty-four were quiet and normal. Three had emesis, but these were all children, from 7 to 11 years of age, who had received rather large preliminary doses of morphine. In several patients an airway was required for a number of hours. It was usually left in until the laryngeal reflex returned. The rectal method of administration was used in all but one of this series of patients, the latter receiving an intravenous injection. The dose for the rectal injection was 0.1 Gm. per kilogram of body weight, or 1.5 cc. of a 3 per cent solution per pound of body weight.

The chief disadvantage and danger of the drug, as pointed out by Dr. Waters who has used it in several hundred cases, is the uncertainty connected with the mass done, which once injected cannot be controlled. The immediate postoperative care is somewhat more complicated, owing to the prolonged sleep. A nurse should be in constant attendance until the patient is conscious, which may be after many hours. I endorse it tentatively for cases in which local anesthesia cannot be employed.

DR. W. H. WILMER: In regard to the question that Dr. Holloway asked, we find that children are not very susceptible to tribromethanol, even when 100 mg. per kilogram of weight is given. In some who seem to be fast asleep, the reflexes can be elicited. Our method has been to test the corneal and deep reflexes, and finally to pinch the conjunctiva with forceps. If there is no response, supplemental ether is not given. One of the weak spots in tribromethanol anesthesia is the fact that there is no way of telling beforehand who is going to be susceptible to it and who is not. The narcosis varies greatly. I operated one day on two children of about the same age. On one, a white child, I performed the operation comfortably without supplemental ether; when the reflexes were tried in the other child, a colored girl perhaps 4 years of age, she said that she could jump off the table and dance the "black bottom," which she did.

There are two conditions in which I think tribromethanol is particularly valuable. We all know the difficulty of removing a cataract when the patient is suffering from exophthalmic goiter. Patients of that type, when given tribromethanol beforehand in their rooms, usually escape all the excitement and the accompanying rise in blood pressure. When the tribromethanol is supplemented by a local anesthetic, the difficulty in operation is much reduced.

The patient who has lost one eye from an explosive hemorrhage is the bête noir of ophthalmic surgeons. Some of these patients suffer from high blood pressure; when tribromethanol is used in addition to a local anesthetic, the feeling that the systolic pressure has probably been reduced by from 40 to 50 mm. of mercury gives the operator a sense of confidence.

REMOVAL OF GLASS FROM THE ANTERIOR CHAMBER. DR. JOHN W. BURKE, Washington, D. C.

Few cases of glass in the anterior chamber have been reported in the literature. There has been difficulty in finding small pieces. A great advance in diagnosis has been made since the use of the slit-lamp. In one case glass had been tolerated for seventeen years without symptoms. Symptoms began only after the glass had pushed through the iris and had become free in the anterior chamber. The first attempt to remove it was unsuccessful; difficulties were encountered, the glass disappearing for six weeks. The second attempt was successful.

DISCUSSION

DR. E. C. ELLETT, Memphis, Tenn.: Dr. Ellett spoke of two cases, one of which had previously been reported (*Am. J. Ophth.* **13**: 49, 1930).

DR. W. E. LAMBERT, New York: In one case I removed a piece of glass with a wire loop.

DR. GEORGE S. DERBY, Boston: Free glass may be removed from the anterior chamber by incision and irrigation. Nonshatterable glasses should be used by children and persons playing such games as baseball, tennis, squash and rackets and in dangerous occupations in which goggles are not used.

DR. EDWARD JACKSON, Denver: It is frequently difficult to find fragments and foreign bodies at the time of the first operation.

DR. JOHN W. BURKE: It is wise to make the corneal incision on a plane with the iris, so that the overhanging scleral lip will not catch the foreign body.

SMALL TUMOR OF THE IRIS WHICH CAUSED SECONDARY GLAUCOMA; RELATION OF MELANOMA TO SARCOMA OF THE IRIS. DR. ARNOLD KNAPP, New York.

This article appears in full in this issue, p. 720.

DISCUSSION

DR. F. H. VERHOEFF, Boston: I believe that a loss of roundness of the pupil is important in these cases. In early ring sarcoma the

growth does not extend all the way around. The relative malignancy of these tumors is not known; this is also true in diffuse sarcoma of the choroid.

Dr. Jonas Friedenwald, Baltimore: The original melanoma showed the characteristic appearance of a benign tumor. The malignant growth overlaid the nonmalignant nevus and was more deeply pigmented. Several small implantation metastases in the iris presented an interesting feature.

Dr. T. B. Holloway, Philadelphia: I observed a case in which one eye had been lost and the other showed a melanoma undergoing sarcomatous degeneration. I resorted to radiation .

Dr. Arnold Knapp: It was impossible to test the motility of the iris in this patient on account of the glaucoma, the pupil being immobile and dilated. The question of this being a beginning ring sarcoma cannot, of course, be answered.

So far as I can learn from the literature reviewed in the paper. these growths occur frequently in three parts. The superficial layer undergoes spindle cell sarcomatous changes which give the characteristics of tumor.

Late Postoperative Separation of the Choroid. Dr. George S. Derby, Boston.

This article appeared in full in the October, 1930, issue of the Archives, p. 530.

DISCUSSION

Dr. W. E. Lambert, New York: In a case that I observed, choroidal detachment was discovered three years after cataract extraction. It subsided spontaneously.

Dr. Arnold Knapp, New York: A number of questions arise in thinking over the condition described in Dr. Derby's paper. There can be no doubt but that there was a leakage of the bleb with the sudden and marked diminution of the tension of the eye. I remember a patient with a trephined eye, living outside of New York, who complained of attacks in which the eye would weep tremendously. These were due to intermittent rupture of the bleb. Fortunately, I was able to cover the defect with a conjunctival flap before serious damage was done.

The question of the cause of choroidal detachment is not as simple as it seems. There are two points that are difficult to explain. The first is the rather common experience that detachment of the choroid does not occur immediately after operation, but after a number of days in which the anterior chamber has more or less regained its normal depth; then suddenly the anterior chamber becomes shallow, and on further examination one finds a detachment of the choroid. Secondly, while compression of the sclera exerts a negative action, why does the exudate usually occur on the sides, rarely below and not above?

Dr. F. H. Verhoeff, Boston: Dr. Knapp brought up the question as to the explanation of postoperative separation of the choroid. It seems to me to be caused by the fact that the intra-ocular pressure, that is, the pressure in the vitreous, has become lower than the pressure of the fluids within the tissues of the choroid and ciliary body. This

permits these fluids to become secreted in greater amount and thus to distend the perichoroidal space. When the separation becomes obvious on ophthalmoscopic examination, it has extended far back. No doubt, small anterior separations occur more frequently than is realized.

An interesting fact is that the separation usually persists for a time after the anterior chamber has been fully reformed. This proves that the vitreous can shrink to at least three fourths of its normal volume and then return to its normal size unaltered.

The fact that the separation goes back when the normal intra-ocular pressure is restored proves conclusively that intra-ocular pressure is normally higher than that of the tissue fluids.

Why the separation should occur laterally and below, but not above, I do not know, but no doubt the explanation of this is mechanical also.

Another interesting fact is that the condition does not occur in all cases of low tension, even if the latter is of long duration. There are several possible explanations of this, but I have insufficient time to discuss them.

In some cases in which the condition persists, scleral puncture is indicated. It is not indicated, of course, if the eye remains soft due to continual leakage of the wound. But in some cases, in spite of closure of the wound, the intra-ocular pressure does not become high enough to push the choroid back. In two such cases I have made it go back by scleral puncture. In one of these I accidentally punctured the choroid also, but no harm resulted, and the patient maintained normal vision until he died, several years later.

DR. W. H. WILDER, Chicago: I have seen choroidal separation frequently, and I believe that there is a hydrolytic peculiarity in these cases, or that the choroidal vessels are more permeable. In one of my cases in which an iridectomy was performed, separation and later reattachment and increased tension developed. A second iridectomy was performed, followed by separation. Ultimately the eye was lost.

DR. GEORGE S. DERBY: According to my recollection, there are only two cases in the literature that showed detachment in four quadrants. As Dr. Knapp said, it usually occurs on the sides; but in the case that I have reported there was a small choroidal detachment below, in addition to the lateral ones.

EXOPHTHALMOS ASSOCIATED WITH DIABETES INSIPIDUS AND LARGE DEFECTS IN THE MEMBRANOUS BONES. Dr. John M. Wheeler, New York.

This article will appear in full in a later issue of the ARCHIVES.

DISCUSSION

DR. PARKER HEATH, Detroit: With Dr. Rowland I have had the opportunity to study two patients with this disease. The first was seen at necropsy in January, 1926, and the second was seen the same year and has recovered from the disease. These two case records, with other considerations, are presented in the Presession Volume and in the Transactions of the Section on Ophthalmology of the American Medical Association containing the papers presented at the Detroit Session in June. Of prime interest in this group is the exophthalmos.

I agree with Dr. Wheeler that the masses in the orbit are probably the main cause of the exophthalmos. At least one case—that of Dietrich—has been reported in which there were no other changes than masses of yellowish kernels in the orbit found at autopsy. But other cases have been recorded. In the necropsy that we had in Detroit the loss of the apex of the orbit, causing easing forward of the macular attachment, was suggestive.

Studies of this disease have shown diffuse changes involving the reticular tissue; peculiar isolated signs, such as exophthalmos, diabetes and syphilis, with occasional involvement of the long bones and many other manifestations, are part of the masquerade in the diffuse disease.

Dr. Rowland has been able to find about thirty cases in the literature reported from the pediatric side, the roentgenologic side, general pathology, etc., while ophthalmologic literature apparently contains no reports of these cases. Rowland correlated this diffuse reticular or lipoid disease in several groups. The variation in the clinical pictures seems to be dependent on: (1) the age of the patient and (2) the type of lipoprotein. The picture varies with the outstanding signs, but fundamentally the fat metabolism is disturbed.

Dr. Wheeler's case is of great interest because it apparently shows retinal changes, and this is rather rare in Schiller-Christian's syndrome. His case may be intermediate between the amaurotic family retinal disease group and the pure Schiller-Christian's syndrome. The association of amaurotic family idiocy and liporeticular disease has been noted by Pick and Bielschowsky.

Study of the reaction of the body tissues to fat disturbances, I think, will lead to a future reclassification of obscure manifestations of deposits in the eye.

Dr. T. B. Holloway, Philadelphia: There is an analogy between these cases and those of craniostenosis . It is possible that some cases of craniostenosis are of this type. Diabetes insipidus, however, is by no means the rule in this condition.

Dr. Jonas Friedenwald, Baltimore: I should like to call attention to one other ophthalmologic condition which may be related to this congenital defect, namely, amaurotic family idiocy. In amaurotic family idiocy there are swollen ganglion cells in the retina and in the brain which contain large amounts of lipoprotein. This fact alone would not relate it to the condition that we are discussing, but a case was reported by Pick and Bielschowsky (Ueber lipoidzellige Splenomegalie [Typus Niemann-Pick] und amaurotische Idiotie, *Klin. Wchnschr.* **6:** 1631, 1927) in which, in association with amaurotic family idiocy, there were the visceral changes of Gaucher's disease. This would seem to form a link between these two types of disease. I think that the suggestion of Dr. Heath is correct: that a more thorough knowledge of the lipoid metabolism of the infant will help to solve the relationship of these remarkable conditions.

Dr. John M. Wheeler: One of the striking things about this syndrome, particularly in connection with the cause of proptosis, is that while one can feel the defects in the cranium, one feels no pulsation through the fibrous tissue which separates the brain from the scalp. Apparently there is no increased intracranial pressure. That makes it

rather unlikely, I think, that the extreme proptosis characterizing these cases can be caused by the weight of the brain on the structures of the orbit, or even by the loss of tissue at the apex. It seems to me that it is much more likely that the principal cause of the exophthalmos is the tumor formation in the orbit.

The condition is strikingly one of infancy. A few cases of this condition, or one similar, have been reported in youths, but most of the cases that truly belong to this classification are reported in babies. It seems to me altogether likely that the origin of the hyperplastic tissue, at least in some of them, is from embryonal cells; that they originate in the region just behind the hypophysis, and so destroy the function of the tuber cinereum and other structures about the infundibulum and stalk of the hypophysis, and that then diabetes insipidus and dysostosis are likely to result. It is also possible that other forms of tumor which originate in this particular location may give this syndrome, may destroy the bone in the region of the hypophysis, may invade the orbit and cause proptosis, and may cause diabetes insipidus and the rest of the syndrome. I think that the condition is essentially one of tumor-like or tumor formation, and that the location of the hyperplastic development is important in the causation of the syndrome.

A Case of Diphtheritic Conjunctivitis. Dr. S. Hanford McKee, Montreal, Canada.

C. B., a girl, aged 8 months, was admitted to the Alexandra Hospital (for contagious diseases) on January 8, with supposed measles. The chief clinical symptom referred to the eyes, which showed a pronounced membrane of the conjunctiva with profuse discharge. Cultures were positive for the Klebs-Löffler bacillus.

DISCUSSION

Dr. F. H. Verhoeff, Boston: I have seen only three or four cases of diphtheria of the eye. I regard the diagnosis from cultures as uncertain; there should always be animal inoculation.

Dr. C. W. Tooker, St. Louis: I observed a case in which the diagnosis was made from cultures.

Dr. C. A. Clapp, Baltimore: I endorse Dr. Verhoeff's stand.

Dr. Burton Chance, Philadelphia: Many years ago, I saw a patient who recovered without corneal involvement.

Dr. C. W. Rutherford, Iowa City: Dr. Rutherford spoke of membranous conjunctivitis, the occasional loss of the eye and the difficulty of determining the etiology.

Dr. S. Hanford McKee: In spite of what has been said, I feel that we are laboring under no delusion in Montreal in respect to the diphtheria bacillus, and that we are able to distinguish it by its staining reactions, morphology and cultural characteristics. There are many types of the diphtheria bacillus. Some of the persons who are working with diphtheria constantly believe that not only can they distinguish this bacillus from a very large group, but that by morphology, staining reactions and culture they can distinguish the virulent from the nonvirulent types. Be that as it may, I have no hesitation in stating in spite of

the absence of animal inoculation tests, that the case presented is unquestionably one of diphtheria of the conjunctiva.

NODULAR OPACITY OF THE CORNEA. DR. EVERETT L. GOAR, Houston, Texas.

Nodular opacity of the cornea was first described by Groenouw in 1890. It is characterized by its familial tendency, bilateral symmetrical distribution and slow progression, with occasional periods of irritation, until useful vision is lost.

The three most plausible theories in regard to the etiology are: (1) It is an hereditary form of degeneration of the superficial lamellae; (2) it is neurotrophic, the degeneration affecting the terminal nerve fibers and endings; (3) it is tuberculous in origin.

The characteristic lesions are in the superficial layers of the corneal stroma and consist of nodules and interlacing lines. The nodules are composed of a substance which probably is hyalin. Sections of the epithelium and superficial lamellae are shown. In the case reported, there was a severe inflammatory reaction with consequent opacification and vascularization of the corneas.

DISCUSSION

DR. ALLEN GREENWOOD, Boston: A mother and three children under my care were all affected with lattice keratitis. In the early stages the lines are much more in evidence than the nodes. Gallemaerts gives an excellent illustration in his book of slit-lamp studies.

DR. JOHN GREEN, St. Louis: Dr. Green referred to his paper on the subject and repeated his conclusions:

"In the present state of our knowledge with regard to this peculiar affection, we must adopt the view that cases of nodular opacity of the cornea, clinically indistinguishable, may differ with respect to their etiology and histology. It seems proper to regard the cases of Groenouw and Fuchs as instances of a general nutritional disturbance of which the sole expression is the corneal lesion. On the other hand, the histologic findings of Wehrli and the diagnostic and therapeutic efficiency of tuberculin in the case here reported, seem to indicate that some of these cases may be, as Wehrli has pointed out, examples of attenuated tuberculosis of the cornea."

DR. CALVIN C. RUSH, Johnstown, Pa.: I saw ten cases in one family. In all instances the center of the area involved was slightly below the center of the cornea, the part most subjected to light. All cases were bilateral and symmetrical. All started when the patient was about 20.

DR. C. A. CLAPP, Baltimore, and DR. BURTON CHANCE, Philadelphia: Dr. Clapp and Dr. Chance also reported cases.

DR. EVERETT L. GOAR: In regard to Dr. Green's belief that grill-like and nodular opacities are definite entities, I doubt that very much. I think that these cases grade from one to the other, and I am certain that this was a mixed case. I think that in most cases of nodular opacity, by examination with the slit-lamp it would be easy to make out definite lines which might be classified as grill-like.

CYSTIC DEGENERATION OF THE RETINA. DR. BERNARD SAMUELS, New York.

This article appeared in full in the October, 1930, issue of the ARCHIVES (p. 476).

DISCUSSION

DR. F. H. VERHOEFF, Boston: While cavities in the retina described by Dr. Samuels have frequently been observed, no one, so far as I know, has hitherto made a systematic study of them. I concur in everything that Dr. Samuels has said except in his statement that the cavities give rise to holes only in the macula. I examined an eye removed by Dr. Derby, in which the separated retina showed four or five of these holes and marked cavity formations in their vicinity. I have several other specimens of this kind in which the holes can be seen both macroscopically and microscopically. It is evident to me that many of the holes seen in separated retinas with the ophthalmoscope are of this nature. I have also found, outside the macula, a hole in the retina without separation of the latter. I think that a hole is especially apt to develop in the macula, but owing to the vastly larger extent of the rest of the retina, holes occur less often in the macula than elsewhere. Since sections of eyes generally are made to pass through the disk and macula, there is more chance of finding a hole in the macula than in the peripheral retina.

When a hole is found in a separated retina, there is always a question as to whether it is the result or the cause of the separation. Cavity-formation is common in separated retinas, and movements of the eye tend to convert these cavities into holes. When I find a hole in a separated retina in older people, I assume that it is the cause of the separation.

DR. JONAS FRIEDENWALD, Baltimore: These extravasations in the retina are particularly interesting in the light that they throw on the anatomic structure of the retina. They occur and spread in those portions of the retina in which the supporting structure is weakest, and for that reason they are most commonly seen in the inner nuclear layer and the layer of Henle, where the supporting structure is most deficient. Those that occur near the disk show on flat section a peculiar pattern of interlacing channels which one would not suspect in looking at a cross-section. The difficulty in seeing them with the ophthalmoscope is due to the fact that the refractive index of the fluid is nearly the same as that of the retina, and that there is therefore little contrast.

DR. GEORGE S. DERBY, Boson: In Vogt's second paper on ignipuncture in retinal separation there are a number of excellent plates which show clinically these spaces in the retina in the process of hole formation (*Klin. Monatsbl. f. Augenh.* **84:** 305 [March] 1930).

DR. BERNARD SAMUELS: I wish to thank those who discussed the paper, especially Dr. Friedenwald for bringing out so well why there is edema in one tissue and cystic formation in another. The optic nerve becomes edematous, and the nerve fibers are separated, but no actual spaces form while the adjacent retina breaks up into lacunae. The optic nerve is the stronger tissue and the retina is the weaker and more delicate, so that under the same influence the latter is torn to pieces, while the former merely becomes edematous.

BENIGN MELANOMA AND MELANO-EPITHELIOMA OF THE CHOROID. DR. H. P. WAGENER and (by invitation) DR. W. L. A. WELLBROCK, Rochester, Minn.

This article appeared in full in the October, 1930, issue of the ARCHIVES, p. 509.

DISCUSSION

DR. F. H. VERHOEFF, Boston: The question as to whether these tumors are epiblastic or mesoblastic in origin probably is of little clinical importance. But to pathologists it has been of great interest and much discussed; this applies also to the pigmented tumors of the skin and the conjunctiva. It seems to me that the essayist has not demonstrated that the choroidal tumor that he found arose from a melanoma of the choroid. It is possible that what he saw in his first clinical observation was actually a beginning sarcoma, not a melanoma. On the other hand, assuming that the tumor did arise at the site of the melanoma, it is not certain that it arose from the cells of the latter. One would suppose that sarcomas often arise from melanomas, but how is one to prove it?

As to the question of the origin of the chromatophores of the choroid, the essayist seems to ignore the fact that all the embryonic evidence goes to show that these cells are mesoblastic in origin. He states that they obtain their pigment from the pigment epithelium. I do not know where he obtained his authority for this statement. The best evidence that we have, the "dopa" reaction of Bloch, shows that the chromatophores of the choroid have the power to produce their own pigment. Bloch contended that these cells therefore should not be called chromatophores. On the other hand, he concluded that the chromatophores of the skin were unable to produce pigment but obtained it from the epithelium of the skin. It is generally conceded that the tumors known as sarcomas of the uvea arise from chromatophores, and it is therefore impossible to escape the conclusion that they are mesoblastic in origin. Many observers, however, believe, as does Bloch, that the pigmented malignant tumors of the skin and conjunctiva are epithelial in origin. But it is a remarkable fact that tumors of the choroid sometimes produce all the different kind of cells found in the tumors of the skin and conjunctiva, so that the question is still open.

DR. GEORGE E. DE SCHWEINITZ, Philadelphia: As Dr. Wagener has referred to some observations of Dr. Shumway and myself, it may be of interest to review them.

The growth was found in the choroid of a colored woman who died following the removal of a sarcoma of the brain. At one point, 3 mm. from the nerve entrance, there was a special thickening of the choroid which was plainly visible to the naked eye. Its long diameter was 1.2 mm. and its greatest width 0.5 mm. There was no evidence of any inflammatory reaction at this point.

This thickening of the choroid was made up of closely massed pigment cells of the usual type of the choroidal stroma cells. Some of them appeared to be slender spindle cells with long processes; others were round or oval with short processes, and there were a number of very large round bodies, likewise densely pigmented, which showed no processes. In addition to these cell forms, pigment was scattered in irregular clumps in the tissue between the unpigmented

cells which made up the rest of the mass. The blood vessels were distended, contained a moderate number of leukocytes and exhibited a decided swelling of their endothelium, but there was no evidence of perivascular new formation.

The use of the ferrocyanide-hydrochloric acid test for iron revealed an interesting condition. The majority of the cells did not stain, but at each end of the growth in exactly the same position in several sections thus treated, a group of the pigmented cells stained a deep blue where they tapered off into the normal choroid. At one point there were two large blood vessels in their vicinity. The bluing could also be seen between the cells, as though there was iron in solution in the tissue, which was being absorbed by the cells.

In the absence of evidences of inflammation, past or present, it seemed proper to consider this choroidal thickening as a melanoma of the choroid in an eye with great increase of the pigment throughout the uveal tract as far as the sections reached.

These observations were made twenty-five years ago. Colored plates exhibiting the structure of the melanoma and the staining of the cells as described were presented.

DR. H. P. WAGENER: As Dr. Verhoeff says, the question whether these tumors are epitheliomas or sarcomas is largely academic. I was under the impression that in the work with the "dopa" reaction, as quoted by Dawson and Knight, it had been demonstrated that the chromatophores of the choroid did not have the power of producing melanin pigment but were only phagocytic for melanin.

Whether the cells can be demonstrated histologically to be epithelial is, as Dr. Verhoeff says, doubtful. Undifferentiated malignant cells of any origin assume varying shapes and types, and it is therefore extremely difficult with undifferentiated cells to demonstrate their epitheliomatous or sarcomatous nature. If one assumes that cells of mesoblastic origin have the power of producing melanin, there is less reason for designating these malignant melanotic tumors of the choroid epitheliomas.

I think that clinically the most important feature of the subject is that one should perhaps look more closely for these melanomatous patches in the eye, that they should be regarded as moles elsewhere in the body—that is, as potentially malignant—and that therefore they should be more carefully watched than they have been in the past.

PRIMARY SARCOMA OF THE IRIS: REMOVAL BY IRIDECTOMY; NO RECURRENCE IN THREE YEARS. DR. WILLIAM ZENTMAYER, Philadelphia.

This article will appear in full in a later issue of the ARCHIVES.

DISCUSSION

DR. E. C. ELLETT, Memphis, Tenn.: I removed a leukosarcoma of the iris by iridectomy. Six months later it recurred locally. The patient refused enucleation, and a year later the growth involved the iris, ciliary body and cornea. The eye was then removed in November, 1928, and to date there has been no recurrence or metastasis.

MONOCHROMATIC VISION. DR. BURTON CHANCE, Philadelphia.

The literature on monochromatic vision was analyzed.

DISCUSSION

DR. JONAS FRIEDENWALD, Baltimore: The following case makes the whole subject even more difficult to understand than it already is. A child whom I first saw when she was 18 months of age had been observed by her mother to be day-blind. She saw well in dim light and could find small objects on the floor of a dimly lit room, but in a brightly lighted room or in the sunlight she stumbled over and bumped against large objects.

Examination of the eyes revealed no ophthalmoscopic anomaly, the pupillary reactions were quite normal and there was a moderate nystagmus of the congenital type. We naturally assumed that she had congenital total color-blindness.

She was last seen a few months before the present meeting, when she was 3½ years of age. I then had my first opportunity to make tests of her color sense. I was much surprised to find that she could distinguish between red and green wool yarns, while she could not distinguish between blue and yellow wool yarns. This curious phenomenon, combined with the day-blindness, is difficult to reconcile with the notion that day-blindness is due to the absence of cones or that the absence of cones necessarily means total color blindness.

DR. E. V. L. BROWN, Chicago: Against the author's citation of an authority who claims that lower animals are devoid of a color sense, I wish to call attention to the experiments of Karl von Frisch of Münich which seem to prove that bees have a well developed color sense for blue and yellow, though not for red.

SECONDARY GLAUCOMA WITH DETACHED RETINA IN BOTH EYES. Dr. William H. Wilder, Chicago.

This article will appear in full in a later issue of the ARCHIVES.

DISCUSSION

DR. BERNARD SAMUELS, New York: It occurs to me that probably the explanation of the good results after the iridencleisis is that a subchoroidal hemorrhage followed, and that as a consequence the choroid was pressed against the retina. With the absorption of the subchoroidal hemorrhage, the choroid may have returned to its normal position, taking the retina with it.

DR. W. H. WILDER: It does not seem to me that that explains it. In the first place, one rarely expects to find increased tension in a case of detachment of the retina. I have never seen a case of increased tension. Such cases have been reported, but they are very infrequent.

SOME OBSERVATIONS IN REGARD TO GLAUCOMA SIMPLEX. DR. HENRY C. HADEN, Houston, Texas.

This article appeared in full in the September, 1930, issue of the ARCHIVES, p. 326.

DISCUSSION

Dr. William Zentmayer, Philadelphia: It is difficult to convince an observer who has seen a glaucomatous cup disappear that on theoretical and anatomic grounds this cannot occur.

I recently read a short paper before the College of Physicians of Philadelphia on the results of sclerocorneal trephining. In one of the cases an interesting feature was the disappearance of the glaucomatous cup. I recently reexamined this man and found no excavation. He had a shallow glaucomatous cup, and at the first examination after the trephine operation the cup had disappeared. Dr. de Schweinitz also saw this patient.

Fuchs stated in his textbook that the glaucomatous excavation may be diagnosed by a difference in the refraction between the margin and the floor of the excavation. He said that this is a valuable procedure, because one can tell whether the excavation is increasing or diminishing. In speaking of Schnabel's cavernous atrophy, he further stated that this cannot be the sole cause of the glaucomatous excavation, because after a decompression operation the excavation has been known to disappear. Dr. Duane made a similar statement. It is true that Fuchs, in a recent paper on "Consequences of Softening of the Eyeball" (Arch. Ophth. 3: 419 [April] 1930) said that when a decompression operation is performed for relief from hypertension the cupping may disappear. He added that this is not due to the lamina cribrosa resuming its normal position, but to the edematous swelling of the tissues left in front of the lamina; when the intra-ocular tension has gone back to its normal level the edema of the papilla disappears and the previous cupping again becomes visible.

I do not feel that this is a contradiction of the previous statement, but that he is really dealing with a different condition.

Dr. Everett L. Goar, Houston, Texas: I endorsed Dr. Haden's assertion that routine tensions should be taken. If this is done, cases of potential glaucoma will occasionally be discovered.

Dr. Walter R. Parker, Detroit: Has Dr. Haden recorded the number of normal patients showing a difference in tension in the two eyes? If this observation has been made, what proportion of his cases show a difference?

Dr. George S. Derby, Boston: I want to congratulate Dr. Haden on his careful work. I agree entirely with what Dr. Goar said: that if the ophthalmologist does his duty to his patients fully he will take the tension in all cases over 40. What is high tension? What is a tension that is abnormal for that particular eye? I think that we all have seen eyes in which glaucomatous changes develop in spite of the fact that they never showed a tension of over 20 according to the Schiötz tonometer. I have had a number of such cases. Then I have seen several cases showing a tension of 30 or more, in which the eye seemed to be able to support that tension. I think that it is difficult to say whether the tension in a given eye is abnormal. Of course a tension from about 25 to 30, according to the old Schiötz tonometer, should excite suspicion.

The difference in the tension of the two eyes seems to me to be significant. I think that the measurement of the visual field is the most important single examination that one can use which will show the progress of a case of glaucoma. As the visual field sometimes fails one, I want to call attention again to the possibility of the light sense examination being of value in some of these cases. These patients will sometimes show a rising level of the light minimum when they will show no changes in the visual field, and of course measurement of the visual field and measurement of the light minimum show definitely the amount of damage that is being done to the eye.

I show the light minimum curves in three cases of glaucoma of the congestive type. In these cases there was no change in the visual fields, although a careful examination was made by quantitative perimetry, but the light minimum curve demonstrated the increasing damage.

DR. ALLEN GREENWOOD, Boston: I want to call attention to the value of Dr. Derby's suggestion in regard to testing the light sense, particularly in patients who have had an operation for glaucoma in one eye and whose other eye is apparently normal, when there is nothing that shows up until changes are found in the light sense. Sometimes in these cases one observes a change in the light sense long before the patient shows the slightest sign of change in the field or in the tension.

DR. PETER C. KRONFELD, Chicago: In connection with the statement made by Dr. Haden and Dr. Wilder a few moments ago, I shall describe a method of detecting the early stages of glaucoma which we have used at the clinic at the University of Chicago for the last two years. The method is very satisfactory, and so far as I know has not failed once in 200 cases.

The method is very simple. We perform punctures of the anterior chamber with a needle and empty the anterior chamber; we then take the tension every twenty to thirty minutes for three or four hours.

A woman, about 55 years of age, had definite simple glaucoma in one eye. We took her to the hospital and took her tension five times a day; it was found to be about 32, 33 or 34, every morning. In the other eye the tension was always below 25. We had her in the hospital for three weeks, and during that time the tension in her other eye was always below 25. We discharged her, but saw her in the outpatient department, and the tension always stayed below 25. We performed a puncture on the eye that showed definite symptoms of glaucoma, and the tension curve dropped to 0, went up to 65 and down to normal in six hours. We did the same thing in the other eye and obtained about the same curve. The tension dropped from 22 to 0, and went up to 62 in two hours. This test has always proved positive in every case of definite glaucoma, and has indicated glaucoma in cases in which five or six months later we were able to see that it was glaucoma. I therefore feel that this behavior of the anterior chamber is typical of simple glaucoma.

The lowest curve of the slide shown indicates the behavior of the normal eye. The other curves show glaucomatous eyes in which we made a diagnosis of glaucoma based on this test. The rate of inflow of fluid is much higher than in the normal eye. Glaucoma cannot be fully explained on the basis of disproportion of outflow. A great deal of increased tension is due to an increase of inflow.

In a case of hydrophthalmos the tension was 65 at the time of puncture; it took three hours to get up to normal, and it took more than six hours to get up to 40. This is not simple glaucoma; it is retention glaucoma, in which practically all the symptoms are due to disturbed outflow. In simple glaucoma most of the disturbance is due to increased inflow.

In the normal eye, the tension is up to 28 and down again in about an hour.

This test should be used in all those cases in which routine tonometric measurements do not show anything, and in cases in which one does not have time to put the decision off for several weeks. When one must decide immediately whether one is dealing with a glaucoma, I think that puncture of the anterior chamber is a very useful test.

DR. H. MAXWELL LANGDON, Philadelphia: How early in the progress of a case in which hypertension had been demonstrated should it be called glaucoma? The general public, as a rule, regards this disease as leading to certain blindness.

DR. PETER C. KRONFELD: We have performed this puncture of the anterior chamber in about thirty-five normal eyes, and in twenty eyes with optic atrophy in tabes; we have used it in many eyes with iridocyclitis and in many eyes with fundus disease. In normal eyes the average behavior has been according to the curve shown on the last chart. By using physostigmine and by bringing up the blood pressure at the time of puncture, we can raise this normal curve from 3 to 5 mm. I remember a case in which we gave a large dose of caffeine a few minutes after the puncture to bring the blood pressure up to 185. The tension curve was about 4 mm. above the average that I have given.

From many other observations I would draw the conclusion that the normal eye, the eye with iridocyclitis, the eye with fundus lesions, the eye with any type of optic atrophy—all those eyes which show no symptoms of simple glaucoma—behave as shown on my last graph, but that by no means can a curve be produced similar to that obtained in simple glaucoma.

SIGHT-SAVING CLASS-WORK FROM THE STANDPOINT OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY AND THE NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS. DR. E. V. L. BROWN, Chicago, and (by invitation) MR. LEWIS H. CARRIS, New York.

Sight-saving classes are being organized in many public schools, always in coöperation with ophthalmologists. There should be agreement among physicians as to procedure.

DISCUSSION

The paper was discussed by Drs. Brown, Green, Wilder, O'Rourke, Holloway, Derby, de Schweinitz, Bruner and Jackson. (See Transactions of the American Ophthalmological Society, 1930.)

MICROSCOPIC OBSERVATIONS IN A CASE OF RETINITIS PIGMENTOSA. DR. F. H. VERHOEFF, Boston.

This article will appear in full in a later issue of the ARCHIVES.

DISCUSSION

DR. JONAS FRIEDENWALD, Baltimore: It may be interesting to add to Dr. Verhoeff's communication the report of a case that I had the opportunity of observing in the past year. A colored woman, aged 35, came to the clinic of the Wilmer Institute, complaining of night-blindness; her eyes showed the typical picture of retinitis pigmentosa. Unfortunately for the certainty of the diagnosis, the patient had a positive Wassermann reaction. A short time after the first examination she died as the result of a cerebral hemorrhage. At autopsy the posterior segments of the globes were obtained for examination. There was no scarring or change in the choroid or any adhesions between the retina and the choroid. In view of this, it seemed to me that the retinal lesion was a true retinitis pigmentosa and could not be attributed to the syphilis. The observations in the retina in large measure coincided with those that Dr. Verhoeff has reported. The rods and cones were almost completely destroyed; a few shrunken distorted cones were found here and there. The nuclei of the rods and cones were almost entirely absent. The inner nuclear layer was well preserved and showed practically no change, but the ganglion cells were almost entirely absent and there was a glial proliferation over the surface of the retina as Dr. Verhoeff has described, and also over the disk.

Most interesting in these sections was the pigment epithelium and the distribution of the pigment in the retina. For the most part the pigment epithelium consisted of a single layer of cells, with only local small areas of increased thickness; however, the amount of pigment and the character of pigment in adjoining areas varied enormously. In some places the pigment cells were almost devoid of pigment, while adjoining epithelial cells contained large, dark, amorphous granules. Nowhere in the pigment epithelium can one find the normal bacillary form of pigment granule that is so characteristic of these cells. In the retina the pigment was contained in cells of which it was impossible to say whether they were cells from the pigment epithelium which had migrated into the retina or whether they were phagocytic cells. These cells were so full of pigment that the nuclei were hidden. These deeply pigmented cells were scattered through the retina, and also accumulated about the blood vessels. In addition to these cells there was some pigment diffusely scattered through the retina, which was not definitely intracellular. Still further, in addition to these deposits of pigment there were some cells in the retina that were apparently normal constituents of the retina which contained pigment.

It is, of course, difficult to identify specific cells in a scarred distorted retina, but it was my impression that these cells which contained pigment were actually normal neuroglial cells of the retina, particularly Müller's fiber cells. This leads to the question whether Müller's fibers may not belong to the class of microglia of Hortega, which are phagocytic glia present in the brain and other nervous tissues. They have migratory characteristics and are thought to be not true glia, but actually reticular cells which are a part of the structure of the brain and have come from the mesoblastic tissue. This speculation has been in part confirmed by some experiments that I performed a number of years ago. I was interested in studying intra-ocular inflammations, and injected trypan

blue into the eyes of some rabbits to study the course of the inflammatory reaction. Trypan blue is a colloid dye that has a marked attraction for the large phagocytic cells. In some of the sections showing the early stage of the reaction I found the dye present in the retina, where it seemed to be particularly taken up by Müller's fibers. In the later stages, after the inflammatory reaction had subsided, the dye was accumulated in the retina in a pattern more or less analogous to that seen in retinitis pigmentosa.

I feel, therefore, that the characteristic pattern of retinitis pigmentosa is due to the anatomy of the retina and to the distribution of the phagocytic cells in the retina, rather than to any essential characteristics of the disease.

DR. T. B. HOLLOWAY, Philadelphia: It has always been a puzzle to me why in certain cases there is excellent capillarity of the nerve-head. In certain cases of retinal degeneration I have seen what might be regarded as a hyperemia of the disk.

DR. F. H. VERHOEFF: If Müller's fibers can take up pigment, it seems to me that in a case of this kind one would see so many of them containing pigment there would be no doubt about the matter, whereas it is difficult to make sure that any of the fibers contain pigment. I have not the slightest doubt but that Müller's fibers are neuroglia cells, because they proliferate and form fibrils which stain differentially in neuroglia stains. This would seem to exclude the possibility of their being mesoblastic in origin as Dr. Friedenwald suggests.

In regard to the optic disk, as I said, it may show little if any atrophy, and in Dr. Holloway's case there was apparently no atrophy at all. Its appearance will vary in different cases, according to the amount of atrophy, the amount of gliosis and the condition of its blood vessels. Owing to the obstruction of the circulation in the retina, the disk no doubt may be congested in some cases.

## THE PERMEABILITY OF THE LENS CAPSULE TO WATER, DEXTROSE AND OTHER SUGAR. DR. JONAS S. FRIEDENWALD, Baltimore.

This article appeared in the September, 1930, issue of the ARCHIVES, p. 350.

### DISCUSSION

DR. F. H. VERHOEFF, Boston: I think that these experiments of Dr. Friedenwald are important and interesting. It does seem probable that senile cataract may be due to decreased permeability of the capsule. On the other hand, this decreased permeability must have a cause, and it is possible that the same cause may produce changes in the lens substance itself. One cannot be perfectly sure, therefore, that the change in the capsule is the cause of the cataract.

Did Dr. Friedenwald determine in his experiments whether or not the epithelium of the capsule was still present, and if so, how much effect it had on the permeability?

It would be interesting to know whether different parts of the capsule have different permeabilities, especially the central anterior and posterior portions and the peripheral portion.

DR. WALTER R. PARKER, Detroit: Is it not possible that the variation of penetrability of the lens capsule may have an important clinical

bearing? For instance, in some cases in which the lens is dislocated into the vitreous, the eye may remain quiet for a long period of time, while in other cases a chemical uveitis may develop in a short time. Is it not possible that resistance of the capsule to absorption in certain cases may protect the eye against uveitis, while in others the the capsule may become absorbed more quickly and the cortical matter exposed, leading to the inflammatory reaction?

DR. PETER C. KRONFELD, Chicago: There is one point that I wish to bring out which seems to confirm Dr. Friedenwald's observations. In my experiments in respiration of the lens I compared the respiration of the lens of a rabbit which was 3 months of age with that of a rabbit aged 1 year. When I was through with the experiment and determined the amount of lactic acid in the lens tissue, I frequently found that in the lens of the older rabbit there was considerable retention of lactic acid, and that the amount of lactic acid around the lens during the experiment did not correspond to the amount found afterward. I therefore think that my experiments confirm Dr. Friedenwald's observations of decreased permeability of the lens capsule in the older animal.

Did he check up on the total sugar count at the end of his experiment? Did he find any decrease of sugar in the entire system during the experiment? In other words, did he notice any amount of destruction or consumption of sugar?

DR. JONAS FRIEDENWALD: In regard to Dr. Kronfeld's last question, I did not make any determination of the total dextrose content in these experiments. The capsule was freed as far as possible from all adherent cells, and it was my experience that in such conditions it remained unchanged when suspended in salt solution and kept in the icebox; for that reason I have not suspected the capsule of having metabolic activity. I am much interested in Dr. Kronfeld's work, which seems to confirm the experiments that I have just reported.

The suggestion of Dr. Parker that differences in the permeability of the capsule in different cases may explain the irregular incidence of uveitis following dislocation of the lens is reasonable. The same explanation could be offered in relation to the uveitis that follows rupture of the capsule in morgagnian cataract. It is my understanding that with the capsule intact no trouble results from morgagnian cataracts, but if the capsule becomes ruptured the toxic products of the disintegrated lens enter the anterior chamber and may cause severe iritis.

The suggestions of Dr. Verhoeff have furnished me with an extra year's work. In the experiments that I have done so far I have studied mainly the anterior part of the capsule, which is more uniform, from one animal to the next, in its thickness and general characteristics than the posterior part. I have not performed any experiments to compare the permeability of the capsule in different places. The technical difficulties would be considerable, because the size of the capsule is limited, and one would have difficulty getting pieces sufficiently large for study.

As to the presence of epithelial cells on the surface of the capsule, the technic reported in a previous paper was used in this experiment. The lens was removed from the eye of the animal, care being taken to avoid traumatization. A cross-shaped incision was made in the center of the posterior surface of the lens, and the nucleus and cortex were

expressed. The capsule was then suspended in salt solution and shaken in a shaking machine for intervals of fifteen minutes so as to remove adherent lens protein and cells. This was repeated with fresh fluid six or seven times until the supernatant fluid contained no demonstrable protein. After such treatment, microscopic examination of the capsule showed no adherent fibers or cells.

Finally, in regard to the possible cause of the decrease in permeability, I have nothing at present to offer. I think the theory of the possible relation of changes in permeability to the etiology of cataract still rests on such a tenuous basis that there is much to do before one reaches the stage of explaining how the changes in permeability are produced. There are, however, several points that may be mentioned: 1. There is an increased thickness of the capsule with age. When one compares the feel of the capsule in a young animal with that of an old animal, one can readily distinguish between the two. The older capsule is thicker, stiffer and tougher. 2. As is well known, the analogous membranes in the eye are Descemet's membrane and the membrane of Bruch. Both of these membranes undergo lipoid degeneration and lipoid infiltration with age. While it has not been possible to demonstrate lipoid in the lens capsule, it seems possible by analogy that lipoid infiltration may take place; such infiltration, I think, would render the capsule less permeable to water-soluble substances. I offer this merely as a possible explanation. At the present time there is no experimental evidence to support it.

### Lamellar Membranes of the Anterior Surface of the Lens.

Dr. T. B. Holloway and Dr. Alfred Cowan, Philadelphia.

The authors refer to the literature of the subject and record three histories that conform to the types described by Elschnig, Kraupa, Vogt, Bedell, Alling, Meyer and others. All three patients were men, aged 60, 64 and 70, respectively. One was a laborer, another a cabinetmaker and the third a puddler and heater in a rolling mill. In one case, bluish-white flake, scale or lacelike changes were noted about the peripheral portion of the anterior surface of the lens, while in the other two cases, transparent, homogeneous, curled membranes were present stretching across the pupillary area.

#### DISCUSSION

Dr. A. N. Alling, New Haven, Conn.: I shall use a slide which Dr. Holloway kindly reproduced for me from a drawing of one of my cases.

In the first place, there is in the center a disk outlined by roughened membrane. On close observation the disk seems a little grayish, slightly different from the zone outside. The outline of the disk is not well shown here, but is actually produced by the folding up and tearing of a membrane which produces protruding tongues. Then outside the disk there is a zone that is practically clear. There are also clusters of little dots that resemble the picture which Dr. Holloway showed, and lastly there are little tags at the edge of the iris. It seems to me that the obvious explanation of this particular type is that the anterior

lamella of the capsule has been torn off by the movement of the iris, that the surface of the lens outside the central disk has been denuded of this friable membrane, and that the dots on the edge of the pupil are tags of the torn-off membrane.

The conclusions that I came to at the time I made these observations are: 1. The membrane is not an exudate. It is too homogeneous, and there are no adhesions of the edge of the pupil to the lens. It would seem rather to be an exfoliation of the anterior lamella of the lens. 2. This is a senile change, although it has been observed in glass-blowers exposed to infra-red rays. 3. Glaucoma is not present as a rule, contrary to Vogt's observation, but there is usually a cataract. Every one of my cases showed a cataract, and only one showed glaucoma. Perhaps I may be obliged to modify my statement in regard to glaucoma, owing to the evidence that has accumulated that glaucoma is often found. I did suggest at the time that it might be caused mechanically by the accumulation in the iris angle of the flakes, for they have even been seen on and attached to the posterior surface of the cornea. The formation of the central disk and its ragged periphery is due to the denuding of the capsule outside of the disk by the movements of the iris; furthermore, the exfoliated lamellae sometimes come off in sheets instead of flakes. In one of my cases the membrane was folded over the pupillary edge.

The question that Dr. Holloway has brought up in regard to the relation of these cases to cataract is one that will require further study.

CHANGES IN REFRACTION WITH PRESBYOPIA. DR. EDWARD JACKSON and (by invitation) DR. DONALD H. O'ROURKE, Denver.

Records of patients over 40 years of age, studied with reference to changes in refraction, especially as to amounts and meridians of astigmatism, show that such changes are common, important and often neglected. Illustrative cases were given.

DISCUSSION

DR. D. H. O'ROURKE, Denver: When we began the tabulation of these case reports, all of which were from our private files, we had no point to prove. Therefore it is of value to disclose the fact that our tabulations and percentage estimations were made independently and separately. When I received by mail Dr. Jackson's percentages in these various groups, it was striking to note that in many instances the figures were exactly like those for my own cases, and that the greatest difference in percentage between Dr. Jackson's and my own in any of the age groups tabulated did not exceed, in any instance, 4 or 5 per cent.

DR. F. H. VERHOEFF, Boston: Did Dr. Jackson analyze a series of these cases to differentiate between changes in the cornea and changes in the lens? I noted that he used the ophthalmometer a good many times, and he therefore may have had sufficient data to make this differentiation.

DR. HENRY C. HADEN, Houston, Texas: It is important to measure the beginning presbyope. I frequently use a cycloplegic for patients up to the age of 50.

Dr. Cassius D. Wescott, Chicago: Dr. Wescott said that he uses homatropine for patients up to the age of 55. He confirmed Dr. Jackson's conclusions.

Dr. Edward Jackson: With reference to the point to which Dr. O'Rourke alluded—the comparison of our statistics, which were tabulated and analyzed independently—as he says, it is only within the last week that either of us has known what the other arrived at. But it would seem worth while to make a sort of double investigation to give this more force, because it is a radical change in our ideas of the later period of life, and it has a practical bearing of great importance.

With reference to the analysis of the corneal and the lens astigmatism brought up by Dr. Verhoeff, the corneal astigmatism, of course, was measured by the ophthalmometer in all cases. We have the data. But we found that there were important things that we could not go into and present the evidence for in any reasonable time. The total astigmatism was always judged by the final correction. The comparison between the cornea and the total astigmatism was based on the data of these two procedures. What we have spoken of as lens astigmatism has also been called supplemental astigmatism, and in some ways that is a good term. But it is not supplemental in the sense that it adds on. The lens astigmatism as a rule enters into the "total astigmatism." Generally, however, the astigmatism that it requires a convex cylinder to correct is less than the astigmatism indicated by the ophthalmometer. That was recognized by those who early used the ophthalmometer. The early statistics from the use of the ophthalmometer were generally obtained from young people, in one instance from more than 700 school children. There has not been the same use of the instrument in later life. The statistics mentioned 400 cases that had been observed for an average of nine years, repeated measurements being made with the ophthalmometer; what they showed was indicated there.

With reference to Dr. Wescott's remarks, I think that we all should recognize that he has been a pioneer in this direction, both as to accurate refraction at all ages and as to periodic health examinations. That is what the examination and refraction of the eye is: a periodic health examination. Periodic health examinations in other directions may be judged as to their importance by what is already known from periodic examination and refraction of the eyes.

The Acuity of Binocular Depth Perception in Hemianopia. Dr. E. V. L. Brown and (by invitation) Dr. Peter C. Kronfeld, Chicago.

This article appears in full in this issue of the Archives, p. 626.

DISCUSSION

Dr. T. B. Holloway, Philadelphia: This interesting paper brings out what I hope may prove to be a serviceable test in cerebral cases with hemaniopia. I do not know what the opinion of the Society is in regard to some of the tests that we have used, but in my hands the Wernicke test has been useless. I think that in a great many of these cases that have been described some lessening in the pupillary reaction was thought

to be present, and it brings up the interesting question as to the Wernicke pupillary reaction: Is there no reaction, or is the reaction lessened? There is exactly the same situation in regard to the Argyll Robertson pupil. Is the Argyll Robertson pupil one such as Argyll Robertson described, a pupil in which no reaction to light occurs, or is it one in which there is lessening of the reaction to light?

I was amazed to hear that Dr. Kronfeld failed to find any preservation of the macular area, if I understood him correctly. What was the size of the test object that he used in making these tests; also what was the amount of illumination on the screen?

Dr. Peter C. Kronfeld, closing: In answer to Dr. Holloway's question, I am not able to give any definite data because of the variety of standards of light under which we examined our patients. We were much interested in the accuracy of fixation under all conditions of light, and we think that between certain limits by increasing the light intensity we accomplished greater accuracy of fixation. That seems to have been the preliminary result of our studies.

Some Ocular Lesions in Septic Diseases: Septic Choroiditis, with Special Reference to the So-Called "Allergic Choroiditis" of Marchesani. Choroiditis and Neuroretinitis in Endocarditis Lenta. Ocular Lesions in Periarteritis Nodosa. Dr. Jonas S. Friedenwald and (by invitation) Dr. Benjamin Rones, Baltimore.

This article will appear in a later issue of the Archives.

DISCUSSION

Dr. Allen Greenwood, Boston: I have nothing to add to this presentation, but it might be of interest that all of the cases of metastatic choroiditis that I saw in France in my travels through the different hospitals occurred when there was a septic infection of a compound fracture of the femur.

Dr. E. V. L. Brown, Chicago: Unless I am mistaken in the Marchesani experiments, there was definite proliferation of the epithelial cells in nodes; it is therefore incorrect to dismiss his experiments as being similar to the author's which show only round cell proliferation.

Dr. Jonas Friedenwald: Dr. Greenwood's suggestion that infected fractures of the long bones are frequently associated with metastatic suppurative choroiditis might be explained by the frequent occurrence of fat emboli in the tissues after fractures. If these emboli are sufficiently large they can lead to the formation of minute infarcts which would be favorable sites for bacterial implantation.

In regard to Dr. Brown's criticism, three points may be urged. 1. Epithelial cells are not conspicuously numerous in Marchesani's illustrations. 2. Occasional epithelial cells are present in our sections. 3. Massive epithelial cell infiltration in the choroid of the rabbit can be produced with the greatest ease with nonspecific inflammatory agents. For instance, I recently examined the eye of a rabbit into which a pure culture of gonococci had been injected. About the colony of gonococci

there was an extensive purulent infiltrate, but the whole choroid was packed with epithelial cells. I do not believe, therefore, that the presence of an occasional epithelial cell in the choroid of a rabbit has any pathognomonic significance.

A Modification of Gonin's Surgical Method of Treatment of Detachment of the Retina. Dr. C. E. Finlay, Havana, Cuba.

This article appears in full in this issue of the Archives, p. 662.

Absorption by Radium of Intra-Ocular Granulation Following a Stab Wound at the Corneal Limbus. Dr. H. Maxwell Langdon, Philadelphia.

A boy, aged 5½ years, was struck in the right eye at the lower limbus by the point of a knife, with a resulting small prolapse of the iris. He was first seen forty-eight hours later with no reaction, clear media and no fundus changes. In one month there was a marked haze over the pupillary area behind the lens, the lens still being clear. Granulomatous tissue seemed to be coming from the wound, and the only thing that seemed to offer any help was radium. Dr. George E. Pfahler was asked to use this. Dr. de Schweinitz and Dr. Wilmer saw the child later and agreed that this was the only treatment to follow.

The vision, which had been good at the first visit, had diminished to the counting of fingers at 2 feet (60.9 cm.).

The radium treatments were started on Sept. 14, 1927; and by October 25, the veil was much thinner and some fundus details could be seen. By Jan. 10, 1928, only a few fibers of the granuloma were left. Unfortunately, there was considerable connective tissue in the retina just below the macular region, but the rest of the fundus seemed healthy. Vision was 5/60. The eye remained quiet. When the patient was last seen, vision was 2/60, and there was a large detachment over the lower portion of the globe. This was first discovered in October, 1928.

Pathologic Changes in a Case of Congenital Hydrophthalmos. Dr. W. Gordon M. Byers, Montreal, Canada.

This article will appear in full in a later issue of the Archives.

A Case of Atypical (Upward) Coloboma of the Choroid. Dr. C. E. Finlay, Havana, Cuba.

This article appears in full in this issue of the Archives, p. 668.

Ocular Hazards and Ocular Safeguards. Dr. Percy Fridenberg, New York.

Dr. Fridenberg commented on some mechanical features of accidents resulting in ocular injuries, and on certain details of ophthalmic function, structure, topography and immunology which seem to have bearing on the incidence, gravity, clinical course or reaction to treatment or to "nature's" healing of these lesions.

## GERMAN OPHTHALMOLOGICAL SOCIETY

PROF. R. SCHEERER, TÜBINGEN, *Reporter*

*Forty-Eighth Meeting, Heidelberg, June 12-14, 1930*

TRANSLATED BY DR. PERCY FRIDENBERG, NEW YORK

*(Klin. Monatsbl. f. Augenh., July, 1930)*
*Continued*

SLIT-LAMP PHOTOGRAPHS. DR. THIEL, Berlin.

The pictures shown, taken with a special apparatus combining illumination and photographic exposure, differ materially from those seen with the corneal microscope. In the usual slit-lamp observation, examination, e. g., of the lens, takes place in successively deeper layers. From the combination of the individual pictures of planes and with the help of stereoscopic vision one gets a solid conception of the object. With photography, on the other hand, only a small area can be presented clearly, namely, to the extent permitted by the depth sharpness (focus) of the objective. As a high light intensity of this lens is essential, the corresponding depth sharpness (Tiefenschärfe) is slight. It follows that in photographs of the lens, the slit-lamp picture of the cornea appears greatly magnified and out of focus. Lens photographs only were demonstrated, as my main purpose was to further, by objective determination of the finer and gross changes in the lens, knowledge of the influence of medication on cataract.

THE TECHNIC OF RED-FREE LIGHT OPHTHALMOSCOPY. DR. A. FRANCESCHETTI and DR. H. K. MUELLER.

Dr. Hartinger of the Zeiss firm has modified the large, simplified Gullstrand instrument for the purpose of examining the fundus with red-free light, using an arc lamp and a red-free filter. The lens reflex is abolished by a diaphragm or blind, but eventually a Hartinger two-part front lens will be used. The following advantages over the Vogt method are noted: a large field of vision, stereoscopic picture, simple manipulation, which offers no difficulty, even to the tyro, in cases of anetropia, astigmatism and nystagmus. The red-free filter can be slipped out, for ophthalmoscopy with ordinary light, and a smoked glass wedge allows the intensity of illumination to be altered so that where Gullstrand's ordinary ophthalmoscope fails on account of opacities in the media, the fundus can still be clearly seen.

THE PATHOGENESIS OF CHOKED DISK. DR. A. BRUECKNER, Basle, Switzerland.

In a patient, aged 61, with atrophy of the right optic nerve dating from childhood, choked disk of the left eye only developed as a result of a (right) cerebellopontile tumor. The atrophic right disk remained unchanged. Histologic examination confirmed the clinical observation: Edema of the optic nerve trunk was found on the nonatrophic side only, as far as the intracranial portion and, to a certain extent, even in the chiasm. Choked disk may be due to various causes, among them a propagated (fortgeleitet) edema of the brain.

A New Photokeratoscope. Dr. H. Hartinger, Jena.

The photokeratoscope, made by Carl Zeiss at the suggestion of Amsler (Lausanne), is intended to photograph the corneal reflex image of a Placido disk or any other geometric figure. A milk glass disk with concentric rings, illuminated from the back, with a special mirror reflex chamber and finder magnifying 5 diopters, is lighted by four lamps connected to the general (Netz) current. A small light, presented for fixation by the patient, is situated at the center of the disk in the focal plane of the photo-objective, which appears to be at infinite distance. The other eye may be excluded and protected from glare by a black disk. The average diameter of the Placido photographs thus obtained was 20 mm. The reflex images extend well beyond the optically significant area of the cornea. The apparatus has a chin rest and also braces the patient's brow to ensure steadiness and a clear picture.

The Vascular System of the Ciliary Processes. Dr. M. Baurmann, Göttingen.

The vascular system of the ciliary processes was compared with that of the choroid on the basis of injections into blood vessels of albino rabbits. The results were presented in a table giving the respective figures of free surface, entire volume, capillary vessel surface, relation of blood volume to organ volume, and average caliber.

The Etiology of Follicular Conjunctivitis. Dr. Lindner and Dr. Rieger, Vienna, Austria.

One of two inoculations into *Macacus rhesus* yielded positive results. In four cases of "folliculosis" a germ was cultured which in characteristics of form and culture was much like Noguchi's *Bacterium granulosis* (sic). Inoculation of two strains in monkeys was negative, but inoculation into Lindner's conjunctivae was followed in about three weeks by the signs of beginning follicular changes in the conjunctiva, without manifest inflammation; two and one-half months later, it had almost entirely disappeared.

A Case of Angiomatosis Retinae. Dr. Junius, Bonn.

A girl, aged 19, had had obscuration and dazzling (Flimmern) of the right eye in 1914. Vision was limited to perception of fingers at a distance of 1 foot (30.5 cm.) peripherally. Tests for a central scotoma were positive. The peripheral field was normal. The superior temporal artery and vein, enormously dilated and tortuous, but still distinguishable, coursed to the structure characteristic of angiomatosis, which in this instance was yellowish red and protruded 5 diopters. The artery made a sharp angular turn on its way to this mass into which it sank. The disk was normal, with slight retinal striation in the immediate neighborhood. A slate gray patch, measuring ⅗ disk diameters, with an ill defined margin, occupied the macular region which was raised ½ diopter and presented a slightly uneven retinal surface, as if traversed by ripples (Rillen) pointing toward the

center of the retina. The finest macular twigs could be followed into this area, but in the last section of their course they appear not as red but as grayish-blue striae. The rest of the fundus was normal, except for insignificant white spots at the nasal side of the disk. In this case there were the early changes at the macula characteristic of angiomatosis retinae. The left eye became blind, and cataractous, but showed no plus tension. The right eye was normal. Careful clinical examination, roentgenograms of the skull and the kidney region and metabolism tests failed to reveal any constitutional abnormality.

PATHOLOGY OF HUMAN CATARACT. DR. J. KUBIK, Prague, Czechoslovakia.

Two hundred and sixty intracapsular cataracts were weighed and the dry content determined after dehydration for twenty-four to forty-eight hours. The absolute weights and the percentage relative to total weight of the lenses were presented in tables showing that the process of maturation and hypermaturation goes hand in hand with a progressive loss of the dry substance. Marked individual variations and the wide range between maxima and minima in one and the same group indicate that the conditions are complex and that judgment in an individual case is not feasible with present knowledge.

DISCUSSION

DR. JESS: As long ago as 1913, I published (*Zeitschrift für Biologie*) similar tables of weights of normal and cataractous ox lenses, showing the same uniform loss of weight in the cataractous lens owing to the diminution of albumin and water content. Studies of human lenses could not be carried out as there were no lenses extracted in the capsule.

THE TYPICAL AND ATYPICAL COURSE OF OCULAR TUBERCULOSIS. DR. E. WERDENBERG, Davos.

In uveal involvement, the three principal forms of disease are characterized histologically (exudation, proliferation, fibrous change) and qualitatively (constitutional symptoms, compensation [sic] or incompensation [sic] of immunity). In tuberculous uveitis there is a marked contrast between the secondary, hypersensitive [sic] form, e. g., secondary, juvenile iridocyclitis, and the two not hypersensitive proliferative and fibrous forms with primary, normal or tertiary diminished sensitivity to toxic action, e. g., in tertiary senile iridocyclitis, in which each separate disease picture has its own typical Ranke immunity reaction. The examination of 500 patients with a tuberculous ocular condition, usually severe, showed, surprisingly, frequent atypical immunity reactions (juvenile uveitis often insensitive to tuberculin; senile uveitis, often hypersensitive; apparent failure of the Ranke system). The explanation for immunity reactions that do not correspond to the disease type is the presence of an increased disturbance of compensation in the defense mechanism, increased toxicity of the general infection and possibly a change in the histologic and serologic (immunity) type in the course of the disease. The interpretation of these phenomena on a biologic basis makes the Ranke system even

more valuable in its application to ocular tuberculosis. There is a further disproportion between the quantity and the quality of the disease process, between the extent of the tissue involvement and the toxicity of the general infection: (1) Ocular and pulmonary (intrathorakal) observation reveals a qualitatively slight, toxic general infection (e. g., in cyclitis, miliary tuberculosis of the choroid), and vice versa (predominately uncompensated or compensated stage); (2) frequent disproportion between the severity of the ocular disease and that of the lung in tuberculosis of the retinal veins. These indications determine the treatment and often the results. The typical form of healing consists in a change from the exudative to the proliferating and fibrous form. In some severe, old cases of tuberculous iridocyclitis with sclerosing keratitis, useful vison was finally attained.

DISCUSSION

Dr. Bartels, Dortmund: My experience corroborates the observation of the hypersensitive course of iridocyclitis in elderly subjects. In one patient, a rather old woman, whose eye was normal before treatment, except for a few "precipates" twenty-four hours after injection of a 1:4 dilution of bacillary emulsion, from twenty to thirty tubercular nodes appeared in the iris. Extraction of the lens when cataract accompanies or follows healed tuberculous iridocyclitis causes marked improvement in some cases.

Dr. Lauber, Vienna, Austria: In many cases of tuberculous iridocyclitis, bacilli are found in the blood that can be cultured directly by Loewenstein's (Vienna) method. It would be interesting and perhaps instructive to determine whether cases with bacillemia run a different course from those in which the blood is free, and whether they react differently to treatment.

Dr. Krueckmann, Berlin: Ranke's law does not apply to the conditions in ocular tuberculosis. Exudative tuberculosis in adolescence may, in spite of initial intensity, run a much more favorable course than tuberculosis in old age, which has a mild beginning. What is the significance of hemorrhages in ocular tuberculosis, especially in the dangerous form of so-called tuberculous periphlebitis during pregnancy? This is perhaps the only instance in which the oculist must decide quickly as to interruption of labor. I have found retinal hemorrhages much more frequently in males than in females.

Dr. Stock, Tübingen: Some cases of iridocyclitis react scarcely at all to roentgen therapy, whereas others improve markedly, although there is no ascertainable clinical difference in the two groups. Possibly the former were not cases of ocular tuberculosis, at all, but cases of some other infection. All chronic iridocyclitis is not tuberculous, and Liebermeister, long ago demonstrated living tubercle bacilli in the blood of patients with comparatively mild general tuberculosis. This disease has never led me to interrupt a pregnancy. Retinal or vitreous hemorrhages are rare in females, and do not seem to have any connection either with menstruation or with pregnancy. I have extracted at least fifty cataracts—all in the capsule—in chronic iridocyclitis, with excellent results; in these cases, however, one must wait until the uveal disease has quieted down.

DR. AXENFELD, Freiburg: 1. Bartel's good results may have been due to better circulation and fluid exchange through a previously obstructed pupil, but it is to be hoped that his communication will not induce any one to remove a cataractous lens with the hope of improving conditions in the presence of a florid tuberculous iridocyclitis. The result would probably be a change not for the better but decidedly for the worse, owing to hemorrhage, increased exudation and connective tissue proliferation. As I said in 1920, these patients must wait, often for years, until the uveal disease has run its course and come to a standstill or until it has been cleared up. Only then can extraction, in the capsule, be of use. 2. Enlarged bronchial glands and a few scattered shadows in the roentgen picture of the lung indicate a pulmonary port of entry of the ocular tuberculosis and so are etiologically significant; progressive, destructive lesions in the lungs (open phthisis), however, with rare exceptions, practically exclude ocular tuberculosis, and vice versa. This interesting and incontrovertible fact may have some relation to "reciprocal" immunity. 3. Interference with, or prevention of, pregnancy in tuberculous iridocyclitis cannot be indiscriminately urged or forbidden. It may be advisable in severe cases of florid inflammation with hemorrhages. In cases that have come to a resting stage, one need not be so severe. The psychic lesion caused by unconditional denial may have more serious results, general and local, than allowing the pregnancy to continue. Continued supervision of the general and ocular condition, however, must be maintained; if the condition becomes worse, the pregnancy may have to be interrupted, after all. One may give conditional assent to marriage, however, if one excludes severe or florid cases. The conditions are quite different from those in phthisis. These patients have no phthisical constitutional habitus that can be bequeathed as a disposition to the offspring; matters are much more favorable in regard to infection of the family or of the following generation.

DR. VON HIPPEL, Göttingen: The possibility that the diagnosis of tuberculosis of the eye is made too frequently led me, years ago, to study histologically a large series of specimens. There was a surprising correspondence between the clinical diagnosis and the pathologic histologic observations. In old, healed, tuberculous iridocyclitis there is a striking disproportion between the marked loss of vision and the apparently normal fundus, probably to be explained in most cases by some concomitant disease of the nerve. Lately, on microscopic examination, I found normal pigment epithelium; the ophthalmoscope would have shown an apparently intact retina, and yet there was complete atrophy (Schwund) of the external retinal layers which explained the loss of vision.

DR. ROHRSCHNEIDER, Berlin: Anterior tuberculosis and hemorrhage into the anterior chamber must also be considered when there is a question of interrupting pregnancy. In one case, there was a change for the worse after operative abortion. In the course of a later pregnancy, after the disease of the eye had markedly improved, operative intervention was the means of preventing the birth of a child at the cost, perhaps, of the mother's sight.

DR. WERDENBERG: Exudative tuberculosis in old age shows marked deviation from the Ranke law. Interruption of pregnancy must be

seriously considered if hemorrhage occurs in both eyes, progresses rapidly in one, or develops after the other eye has been lost. Careful watching, repeated observations, and rapid decision in case of a turn for the worse in the fundus are essential if the moment for operative intervention is not to be missed. Extraction of cataract (Bartels) may be followed by good results if one waits, sometimes a number of years, for the ocular tuberculosis to run its course. Otherwise, intervention must be limited to the most sparing (schonendsten) emergency operations, e. g., for glaucoma. The disproportion between ocular and pulmonary observations in tuberculosis of the retinal veins was demonstrated (Axenfeld): (1) extensive retinal vascular disease of high degree with minimal intrathoracic lesions, and (2) nonprogressive (stationaer) benign [sic] hemorrhages in phthisis with cavities. Roentgen therapy (Stock) is of great value in productive tuberculous uveitis, as proved in 400 of my treatments. In exudative forms, irradiation is a "different" therapy which, even with small doses, may be followed by a severe early reaction.

THE FINER STRUCTURE OF OCULAR TISSUES. DR. E. HERTEL, Leipzig.

Roentgen diagrams (Debye-Scherrer) were presented for the study of "submicroscopic" conditions as indicated by interference phenomena in the cornea, sclera, lens and vitreous. Dehydration with alcohol or after exposure to air was followed by a change in the interference rings of the cornea and sclera, but not of the lens; colloid imbibition was shown in the former alone, and there was a disturbance of the crystalline structure by intramicellular imbibition in the fresh state. The lens contains micelles free from imbibition, even in the fresh condition. Differences in submicroscopic structure also explain the different tensile strengths (Festigkeit) of various tissues. Dried lens matter broke down or could easily be reduced to powder in a mortar, while desiccated cornea and sclera seemed to become tougher and were broken down only with much difficulty. Although fiber diagrams were but faintly indicated, the disappearance of the interference phenomena in the ground-up scleral and corneal tissues, and their presence in the broken-down lens masses seem to show that the micelle "principal chains valence" are more regularly arranged in the former tissues, and that there is more marked matting (Verfilzung) in the latter. This is in keeping with the fact that, according to Ebner, the uniaxial double refraction of the cornea is positive, and that of the lens is negative. Fresh vitreous, like fluids, showed the picture of amorphous substances, but the diagram of the dried powdered substance showed numerous equally distributed shadowy spots which indicated small crystals varying in size and irregularly located.

AN ATTEMPT TO DETERMINE THE OCULAR ENERGY-METABOLISM (WECHSEL). DR. F. P. FISCHER, Leipzig.

Bang's weighed papers were used to determine by Kaneko's method the amount of blood flowing out of a vorticose vein in a unit of time, and thus to calculate the entire amount which streams through the eye in a day, i. e., only 8 cc.; improved technic, however, brought the figures to 40 cc., the same as for the kidney and the narcotized brain of rabbits.

One must, furthermore, determine the oxygen content of both arterial and venous blood and its consumption. This is found to be twice that used up in cerebral "respiration." Carbonic acid determination in arterial and venous blood gave a respiratory quotient of 1.0, invariably, indicating that the eye must metabolize carbohydrates. The data of experiments on rabbits' eyes with figures reduced to correspond with conditions in the human eye showed that the energy requirements of the eye are, on the average, the same as those of other body tissues. In this energy economy the lens seems to play the principal rôle.

ANALYSIS OF THE BLOOD GASES IN THE EYE OF THE RABBIT. DR. MEESMAN, Berlin.

A comparison of the oxygen content of the carotid and the vorticose veins showed that the oxygen consumption of the dark-adapted eye is about equal to that of muscle, when at rest. It is affected by radiant energy and by short wave ultra-red, as well as visible, light, especially the latter.

BALLISTIC ELASTOMETRY AND THE EYE. DR. VOGELSANG, Bonn.

With Magnold's tonometer, a point on the sclera is weighed with 4 Gm. The depth of impression increases for ten seconds, and return to a position of rest takes place after the weight is removed. With an even intra-ocular tension, changes are found in the elasticity of the wall of the eye, as a result of the local action of potassium iodide, caustic potassium and citric acid on the tissues. The ballistic method is superior to the static. The shorter period of oscillation (Schwingung) does away with subsequent elastic stretching and other disturbing reactions in the tissues. There are interesting relations between this procedure and the registration of intra-ocular blood pressure, of the corneal pulse and of the pulsatory variations of the globe and its walls. This study may throw more light on glaucoma.

THE SIGNIFICANCE OF LIPOIDS IN PRIMARY GLAUCOMA. DR. A. PASSOW, Munich.

Hypercholesterinemia was found in thirty patients with glaucoma. Overfeeding rabbits and cats with cholesterol and viosterol produced fatty and chalky degeneration of the blood vessels. The influence of these lesions on fluid exchange in the eye was studied by means of fluorescein and albumin determinations in the fluid of the anterior chamber. Experiments were also made with bengal pink ("rose bengale," dichlortetraiodofluorescein) which appears particularly diffusible into the anterior chamber in weak concentration. On faradic irritation of the cervical sympathetic, Wessely found a diminished penetration of fluorescein and albumin into the interior of the eye, due to vasoconstriction. The vascular degeneration induced by viosterol and bengal pink produces a similar reaction, viz., vasoconstriction, which is absent in the case of fluorescein. In the rabbits fed cholesterol, the larger vessels, such as the aorta, showed marked fatty degeneration of the intima and media. In the eye there was marked storage of lipoids, especially at the sclerocorneal margin and in the ciliary body and proc-

esses. In the rabbits given viosterol, the aorta was almost completely degenerated with chalky changes extending via the carotid to the ocular vessels. There was a suggestion of chalky degeneration in the walls of the vessels of the iris and ciliary body. In the organs especially disposed to calcification (the lung, stomach, heart and especially the kidneys), the changes were most marked. Besides the sclerosing effect on the vessels, cholesterol and viosterol reduced tension in animal eyes and inhibited the diffusion of substances from the blood into the fluid of the anterior chamber. Lipoids and allied bodies do not seem to have any significance in the pathogenesis of glaucoma.

MORPHOLOGIC CHANGES IN PHARMACOTOXIC OCULAR REACTIONS. DR. F. POOS, Münster.

Epinephrine, insulin, dionin and histamine, a single large dose of which causes hypotony lasting for a considerable time, lead to a general toxic damaging of the capillaries of the uveal tract, with sequelae resembling inflammation. After a subconjunctival injection, the process begins acutely with a dense, round cell infiltration corresponding to the diffusion of the poison in the globe and extending far back. Marked dilatation of the vessels and edema are noted in many places. The tissue spaces of the sclera are crowded with leukocytes which migrate into the ciliary processes and into the anterior segment of the choroid. In the anterior chamber one finds coagula containing lymphocytes and leukocytes which accumulate, particularly in the lower filtration angle, and which may completely block the tissue bays (Gewebsbuchten). The ciliary body shows changes that can be explained only as the result of direct and marked toxic damage to capillaries by the poisons diffusing within the interior of the eyes. These changes consist in the formation of edema of varying intensity in various places, large Greef cysts, enormous dilatation of vessels, which here and there are stuffed with red blood cells (regional stasis), and emigration (diapedesis) of white blood cells, which occasionally form dense perivascular infiltrates. In the choroid, all sections showed a definite increase of lymphocytes and sometimes circumscribed, denser infiltrates. The same tissue changes, but less marked, are found if dionin powder, epinephrine or histamine in the form of Hamburger's epinephrine substitutes, glaucosan and amino-glaucosan, are introduced into the conjunctival sac. The process is one of "selective" damage to the cells of the wall of the capillaries bearing the earmarks of a true inflammation underlying the "reactive hyperemia" which accompanies the protracted hypotony.

THE RELATION BETWEEN FLUID METABOLISM AND GLAUCOMA. DR. W. WEGNER, Greifswald.

Drinking tests in twenty-five patients with primary glaucoma were inconclusive. Of thirty-six suitable eyes with undoubted glaucoma simplex, twenty-three responded to the ingestion of large amounts of water with an increase of tension of from 3 to 20 mm. of mercury. This did not seem to have any ascertainable connection with a thinning

of the blood from fluid intake. Glaucoma does not cause any definite typical pathologic alteration of the plasma, and vice versa. The intra-ocular capillaries do not mirror the condition of the terminal vascular branches in the general circulation. They seem to have a significant independence in the organism. Four cases of uncompensated glaucoma showed a marked disturbance of fluid metabolism (Wasserhaushalt); one case of hydrophthalmus gave a normal response to the drinking test.

GLAUCOMA WITHOUT HIGH TENSION. DR. THIEL, Berlin.

There is no absolute parallelism between the depth of the excavation of the nerve head and the degree of intra-ocular hypertension. Elschnig called those cases of optic nerve atrophy with deep cupping to the margin but without plus tension "glaucoma without high tension," as it is his opinion that every "total" excavation is of glaucomatous origin. Schmidt-Rimpler, Glauning, Caspar and Roenne claim that this condition may be caused by a descending atrophy of the optic nerve. The intracranial portion is held in place almost immovably by topographic relations in the middle fossa, so that lesions in the neighboring organs, especially the pituitary and the internal carotid (circle of Willis), easily cause damage mechanically. Tumor or syphilitic neoplastic tissue at the base or of the hypophysis may produce this fundus picture. In other cases, the deep total excavation without plus tension was due to demonstrable sclerosis of the internal carotid, the circle of Willis and its collateral branches. Roentgenograms will show these changes. Dorso-anterior plates project the shadow of the carotid into the superior orbital fissure. Even better pictures are presented with bitemporal exposure, as the shadow of the chalky carotid appears directly in the center of the sella turcica, i. e., where ordinarily there is no bone shadow. Stereoscopic roentgenography is essential to prove the extrasellar location of any such shadow. In these cases one finds characteristic field defects, central scotoma, and enlargement of the blind spot, as noted by Wilbrand, Saenger, Fuchs, Horniker and others. The histopathology of total excavation with plus tension includes direct injury to the nerve fibers by pressure on the sclerosed internal carotid and the formation of areas of softening and degeneration by partial or complete occlusion of the similarly damaged nutrient blood vessels in the nerve or sheaths (Fuchs, von Stier). If these zones of softening are located immediately back of the lamina cribrosa, the latter may give way, and, receding, produce total, pseudoglaucomatous cupping (vom Hofe). In the cases due to tumor of the base, there is probably a true pressure atrophy. This may accentuate a deep or wide physiologic excavation (Schweigger, Knies). Possibly, an unusually deep-seated lamina may play a rôle in some cases. Some peculiar anomaly in the structure of the papilla is essential to explain the total cupping in these cases and their rarity considering the frequency of atrophy of the optic nerve. One had better not use the term "glaucoma without high tension" as these cases with progressive loss of vision, in spite of pilocarpine or hypotonizing operations such as trephining, are not cases of real glaucoma although there is total excavation.

EXPERIMENTAL ANALYSIS OF HYPOTONY PRODUCED BY ERYTHROPHLEIN. DR. K. W. ASCHER, Prague, Czechoslovakia.

Slit-lamp examination shows that there is always an increase of from 40 to 180 per cent in the thickness and weight of the cornea after the instillation or subconjunctival injection of this medicament. Water and a 9 per cent salt solution also cause an increase in weight, although less marked, as does philocarpine. Atrophine causes a slight decrease. Dehydration tests showed that the increase is due almost entirely to water absorption by the tissues of the wall of the globe, especially of the cornea, with slight diminution of volume and a decided diminution of water absorption in the interior; this explains the action of erythrophlein in reducing tension.

MEASUREMENT OF INTRA-OCULAR TENSION. DR. H. K. MUELLER, Basle, Switzerland.

Knowledge of the range of physiologically normal limits of ocular tension is still uncertain. The proof of the correctness of maxima and minima can only be afforded by calculations based on "variational" statistics. Thus, Gjessing analyzed the tonometric figures for 2,000 normal eyes on the basis of the theory of probabilities. Using the Schiötz tonometer and the 5.5 weight, all physiologically normal [sic], tensions are found between indicator excursions from 2.2 to 8, i. e., from 33.5 to 18 mm. of mercury (Schiötz). The eyes of fifty cadavers were tested, in situ, to determine the average error of the tonometer. This amounts to $\pm 5.5$ mm. of mercury with the 5.5 weight. With increasing age, the physiologically normal intra-ocular tension decreases, although the blood pressure increases. Age graphs reduce the average error to 4 mm. This remnant depends on mechanical imperfections of the tonometer and on individual variations in the physical characteristics of the ocular coverings which persist in spite of allowance for age factors. Ballistic tonometric tests were carried out, in which a small pendulum was allowed to fall against the eye of the cadaver and the depth of impression was recorded by photography after artificially inducing various definite states of intra-ocular tension by manometric methods. The average error of the ballistic method was found to be from 3 to 3.5 mm.

THE ACTION OF SMALL AMOUNTS (FROM 0.05 TO 0.1 CC.) OF PHYSIOLOGIC SALT SOLUTION INJECTED INTO THE VITREOUS. DR. SCHMELZER, Erlangen.

Lowering of tension is marked and prompt but gives way to normal conditions unless the eye has been subjected to repeated weighting in the course of tonometric tests. The more lasting hypotony then observed is due in part to the puncturing of the globe, and in part to repeated weighting which probably forces a small amount of the fluid out of the eye with each application of the testing instrument, thus causing a sort of puncture-irritation (Punktionsreiz).

DISCUSSION

DR. LOBECK, Heidelberg: I can corroborate Wegner's conclusion as to the negative result of the drinking tests in glaucoma. This indi-

cates that so far it has not been proved that there is invariably some lesion of the capillary endothelial cells in glaucoma which is a factor in the pathogenesis.

Dr. Onken, Wilhelmshaven: A man, aged 48, had lost one eye by injury, several years before examination. There was sudden and marked loss of vision in the previously normal eye. Embolism of the macular arteries, central retinal edema, a cherry-red spot and a large central scotoma were found. After several weeks, small hemorrhages were noted in the inner retinal layers, with the gradual formation of vascular anastomoses on the disk. There was absorption of the hemorrhages, followed in a few weeks by the sudden and rapidly progressive development of a "total" glaucomatous cup, 5 diopters deep. The hemorrhages disappeared completely. The condition unchanged for a year. The tension was from 30 to 35 mm. of mercury; it was not appreciably alterable. Examination of the field showed that the central scotoma persisted. There were no other changes. The pressure of edema on the central retinal vein which caused the hemorrhage may have resulted in areas of softening and atrophy in the nerve head directly back of the lamina, causing recession and the appearance of a total (pseudoglaucomatous) excavation which could not have been due to the very slight rise in intra-ocular tension. In fact, it would seem more probable that the latter was caused by the disturbance of circulation in the posterior segment of the eye.

Dr. Fischer, Leipzig: I found changes in intensity and in the form of water-binding in increased intra-ocular tension, in fifty human eyes examined for the intensity and capacity of absorption of fluid by the outer coats of the eye.

Dr. Marchesani, Munich: A white disk with excavation (Thiel) does not necessarily indicate histologic atrophy, i. e., an actual diminution in the size or loss of nerve fibers. We find on autopsy in extremely aged persons, especially in advanced arteriosclerosis (not hypertension) there is a shrinking in volume of the brain with marked convolutions and deep sulci. This is due to a diminished water-binding capacity of the brain. As a part of this structure, the optic nerve may well show the same hydrophysical biologic characteristic. In the cat the injection of distilled water into one carotid and of 10 per cent salt solution into the other—of course, under equal conditions of pressure—causes swelling of one half of the brain and shriveling of the other. There is relative edema of one disk and relative excavation of the other.

Dr. Wessely: In spite of Mueller's careful research on error averages at various ages, in tonometry, one should not attach too much importance to the actual figures obtained. The relative changes in one and the same eye are decidedly more important. In tests on the eyes of cadavers one must consider, besides the age of the subject, the length of time that has elapsed since death, as changes take place in the eyes post mortem which soon alter conditions of elasticity.

Dr. vom Hofe, Leipzig: I used one drop of 5 per cent cocaine to prevent pain in injection and to keep the animals quiet. After this anesthesia wore off, the subsequent measurements were always made under phenacaine anesthesia. The provisional increase of tension after injection into the vitreous lasts about fifteen minutes. If vitreous fluid

actually escapes through the minute, rapidly occluding needle puncture, a slight bulging of the conjunctiva is noted and tension sinks immediately. In the experiments submitted, however, the pressure tension sank gradually from immeasurable heights above the values of origin (Ausgangs werthe) to depths that varied in individual cases. The continuation of this hypotony varies also. Cases in which vitreous escaped were not included in my series. Repeated tonometric measurements were made only in the first hour in order to obtain a correct impression of the time conditions of the early changes in tension. On the following days, only one tension value was determined. Rossin, too, noted lowered tension after the injection of sodium chloride. This probably induces some change in the vitreous and intra-ocular vessels as well as in the coats of the eye, as indicated by a fleeting haziness of the cornea which comes on at the moment of injection and accompanies the primary rise of tension caused by it. This certainly indicates some tissue change. It would be surprising if this had no influence on intra-ocular tension.

DR. SCHREIBER, Heidelberg: It would be interesting to know whether in any of Thiel's cases with atrophic excavation the fundus picture, and especially the disk, had been seen before atrophy? A pre-existing physiologic excavation is a prerequisite for an atrophic cup. The atrophic process alone leads merely to a saucer-like flattening, never to cupping of the disk. I fail to understand how the first two pictures projected by Thiel, with total pot-shaped (Kessel) excavation could be explained histologically as atrophic.

DR. POOS, Münster: Ascher's observation of increase of weight in the ocular walls after subconjunctival injection is due, at least in the case of epinephrine, not only to simple imbibition of fluid, but to inflammatory infiltration, especially of the sclera. We all agree with Schmelzer that repeated tonometry reduces tension by a sort of massage effect, but long continued and marked hypotony must depend on intra-ocular reactions. The injection of 0.2 cc. of physiologic solution of sodium chloride, without puncture of the anterior chamber, is accomplished by marked stretching (Ueberdehnung) of the cornea, indicated by transient haziness, which is comparable to the lesion produced by bruising (Prellung). Histologic examination of such an eye, several days later, shows edematous changes in the ciliary body, and the escape of white blood cells into the vitreous. The globe requires several days in which to recover from this injury by blunt violence; meanwhile it remains abnormally soft.

DR. THIEL: I have always accepted a large, saucer-like physiologic excavation as a prerequisite for the development of total (randstaendig) cupping. In one case of unilaterally progressive tumor of the hypophysis I was able to follow the gradual extension of the preexisting saucer-like excavation in every direction toward the disk margin in the corresponding eye (Projection). Senile tissue atrophy (Marchesani) undoubtedly plays a rôle in the pathogenesis of deep "total" excavation, but there is also, beyond doubt, an atrophic process in the nerve in the cases of carotid sclerosis, as shown by the field defects and the progressive loss of vision.

DR. ASCHER, Prague, Czechoslovakia: An instrument would be of great value which would allow one to subtract the factor of corneal elasticity from tonometric reading and so determine changes in resistance, not only from one eye to another, but at various points of the same cornea (scars, ectasiae, etc.). Four years before the present meeting, I called the attention of this society to the relation between fluid-economy (Wasserhaushalt) and intra-ocular tension after insulin therapy in diabetes. Of course, imbibition alone does not explain the total increase of weight (Poos) but it is the principal factor. Cell proliferation could not explain an increase of 20 per cent or even 50 per cent in the weight of the cornea in a single day, and the loss of weight after atropine can hardly be explained except on the basis of loss of fluid.

DR. SCHMELZER: Of course (vom Hofe), tonometric tests of both rabbit eyes were made daily for about fourteen days after the injections of saline solution. Persistent hypotony was noted, however, only in those cases in which the tonometer had been applied repeatedly at short intervals. It will occur with intra-ocular tissue changes (Poos) after the injection of a somewhat larger quantity (0.2 cc.) into the vitreous, but only secondarily as a result of intra-ocular lesions which may be due indirectly to the marked and persistent initial rise in tension. I, too, noted a certain amount of clouding of the disk on the day of injection, which I could not explain.

STUDIES OF THE AQUEOUS HUMOR. DR. K. VELHAGEN, Halle.

Tests of the autonomic innervation of the eye were performed on cats in ether narcosis with unilateral faradization of either the cervical sympathetic or the ciliary ganglion. No "sympatheticomimetic" (Loewi) substance appeared in the fluid of the anterior chamber (the test material included isolated frog heart, rabbit intestine, frog pupil, etc.), but two phenomena suggested the presence of a parasympatheticotonic substance, viz., fluid from the anterior chamber of an eye, the ciliary ganglion of which has been stimulated, causes a slight contraction of the pupil of a cat when injected subconjunctivally; it also causes agglutination (Ballen) of the melanophores of the iris of frogs. These properties characterize acetylcholine, which may be identical with some vagus substance (Loewi). Calcium and albumin contents are lower in the sympatheticotonic and higher in the vagotonic eye than in the uninfluenced eye. As calcium contracts the pupil and agglutinates melanophores of cold-blooded animals, it appears possible that the phenomena noted may be due to calcium transpositions (Verschiebungen) according to Zondek's theory, or, more probably are due to the alteration of albumin content as a central mechanism [sic], although this does not explain, for instance, the quite different action of serum, which is still rich in albumin and calcium, on the melanophores of cold-blooded animals.

AGGLUTININS OF THE AQUEOUS. DR. MYLIUS, Hamburg.

Tests were performed to determine the possible existence of individual serologic differences in the composition of the fluid of the anterior chamber analogous to those uniformly found in the blood and in most

organs hitherto examined. Further investigation included the differentiation of hetero-hemagglutinins appearing in the fluid of the anterior chamber of rabbits, and a study of the production and the further differentiation of isohemagglutinins in man. The (late) appearance, in the anterior chamber of normal and immunized rabbits, of agglutinins in response to the injection of red blood cells of human beings is not species-specific. One always finds a number of group-specific partial agglutinins that correspond to the constitutionally fundamental (angelegt) agglutinins present in the plasma. In the first anterior chamber fluid of immunized rabbits withdrawn, only agglutinins against the erythrocytes of the particular blood group used for immunizing were found. Normal anterior chamber fluid does not show individual serobiologic characteristics like those of the blood, but these may be acquired under the influence of factors that are conditional in the widest sense, such as inflammation. Consideration of blood group relationship of donor and receiver may be advisable or necessary in case of corneal transplantation or substitution of vitreous.

RESORPTION FROM THE ANTERIOR CHAMBER. DR. K. SONDERMANN, Berlin.

A man, aged 79, had glaucoma simplex and corneal ulcer. Iridectomy was followed by hemorrhage into the anterior chamber. Death occurred from intercurrent disease one week later at which time the hyphemia had not been absorbed. On histologic examination the paths for this process proved to be those found by Nuel and Benoit by injections of india ink into the vitreous, viz., the ligamentum pectinatum, Schlemm's canal, Fontana's spaces, the ciliary body and the iris. In resorption via the ligamentum pectinatum, the blood cells enter the medially located portion of the framework and then proceed (verlaufen) diagonally and laterally to Schlemm's canal, and generally to the point at which I have determined the presence of a small inner canal which is in open communication with the spaces of the ligamentum pectinatum. At some points, blood cells are found lying in the interior "canaliculi" and more or less numerously in Schlemm's canal itself. In Fontana's spaces and the ciliary body the blood cells are found, in part, arranged in rows, obviously due to their being contained in lymph spaces. The points of entrance in the iris are exclusively the crypts present at the pupil and near the root. In the iris itself occasional blood cells are found singly or in smaller or larger accumulations irregularly scattered in the tissue. Their further elimination has so far not been determined. On the surface and to some extent in the interior of tissues bordering on the filtration angle, conglomerates of red blood cells were found, the appearance of which suggested large migratory cells, the so-called macrophages, which had ingested the red blood cells and then caused them to disorganize. Intracellular rust-brown pigment particles, evidently caused by the breaking down of red cells, gave no iron reaction. The hematogenous nature of this pigment, however, must not be absolutely denied, as chemical reactions of a hitherto unknown nature may play a rôle.

AN APPARATUS FOR STEREOSCOPIC PHOTOGRAPHY WITH RED-FREE FOCAL AND DIFFUSE LIGHT. DR. THORNER, Berlin.

Dr. Thorner reviews all the reflex-free ophthalmoscopes, back to his own instrument (International Ophthalmological Congress, Utrecht, 1899). His stereoscopic ophthalmoscope, which casts an image from a concave mirror without central reflex, is especially suitable for use with red-free light. By means of a bulb with a bright metallic filament, it shows clearly the yellow color of the macula and the other fine details of the retinal picture. For focal illumination, the image of a very bright, fine, vertical line is thrown on the retina and viewed steroscopically. The slightest differences in level can be noted, as in Koeppe's method, but with the greatest ease (Emil Busch Company, Rathenow).

*(To be continued)*

---

## ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

H. DICKINSON, *Reporter*

*June 13, 1930*

CYRIL H. WALKER, F.R.C.S., *President, in the Chair*

AN EXPERIMENT IN KERATOPLASTY. MR. J. W. TUDOR THOMAS.

Mr. Thomas said that at the termination of four years of experimental work in efforts to carry out transplantations on rabbits' corneas the results he had achieved were so poor that it seemed hopeless to expect to obtain transparent grafts, such as he did secure at a later date. The latter he demonstrated later at the Annual Congress of the Ophthalmological Society.

The purpose of the experiment described was to ascertain whether the retention of a very minute pedicle of corneal tissue would suffice to enable the graft to maintain its transparency. He made a rectangular incision into the cornea of a rabbit, the whole corneal thickness being penetrated, but a pedicle 2 mm. thick was retained at the posterosuperior cornea. The sides of the rectangle measured 7.5 by 4 mm., and the incision was across the center of the cornea. Four stitches secured the flap in position. There was good union of the flap, and, with the exception of a marginal scar, the flap later became transparent. It was in this way shown that the keeping of a small pedicle enabled the graft to maintain its transparency. Though there was only a pedicle 2 mm. in width, it sufficed to retain life in the graft, without the loss of transparency. The graft was cloudy for a week after the operation, and at the end of a fortnight was still slightly cloudy. After three weeks the graft had become sensitive to touch over an area embraced from the 2 mm. pedicle to the posterior 2.5 mm. of the inferior margin. At this time the graft was clear in the sensitive area, also somewhat beyond it, the rest of the graft being only slightly cloudy. At the end

of a month the graft was clear except in the case of the marginal scar. Thirteen weeks from the date of the operation sensation was normal all over the scar, except for about a square millimeter near the middle of the anterior end of the graft. Twenty-eight months after the operation the animal was killed, and microscopic examination of the sections showed that in general the graft was a little thicker than the remainder of the cornea, and that Descemet's membrane was at least as thick as the normal membrane; also it stained a little deeper. In the main graft there was no appreciable increase in the number of nuclei; these sections were demonstrated on the screen. But at the opaque margins of the graft, the following changes were evident under the microscope. The epithelium was thickened and there were six layers of cells, the basal ones being elongated. In this region the stroma contained twice as many nuclei, and there were numerous spindle-shaped spaces between the fibers. Between the free end of Descemet's membrane in the substance of the cornea and the posterior membrane the tissue fibers were coarser and more numerous, some iris pigment could be seen at the site of the anterior synechiae, and the pigmented cells were covered by a layer of endothelial cells with a new Descemet's membrane. A minute split was discovered between the two layers of Descemet's membrane; in places this split could be seen to extend throughout the whole breadth of the graft. A small detached portion of Descemet's membrane could be seen curled up in the region of the scar near the posterior surface, but covered posteriorly by the new membrane. This showed that detached portions of Descemet's membrane were but little altered in the course of years. The appearances of Descemet's membrane in this case agreed with the observations of others as to the healing of corneal wounds, namely, that the cut ends of the membrane did not unite, but were joined by the new membrane which was formed, either to unite the cut ends or to fuse with the posterior surface. In this case a new membrane had been formed, which passed across the graft and fused almost completely with the graft's original Descemet's membrane. It seemed clear that the marginal portion of the new membrane must have been formed by the endothelial cells of the surrounding cornea. The specimen showed that a new membrane could be formed under such conditions as obtained in this experiment, over quite a large area, and that it actually covered and fused with the membrane of the graft. It did not accord with the view expressed by Weinstein that the new membrane was composed of differentiated corneal lamellae. The experiment showed that the retention of even a small pedicle of corneal tissue was a material help in revitalizing the graft. Mr. Thomas' experimental experience had been that if a small pedicle had not been retained, even the same method of stitching as in the present case would have produced an opaque graft, and if the present graft had been left without stitches to secure it, the eye would probably have been useless, or at best there would have been a staphylomatous cornea.

If in man a clean perforating wound of the cornea has been sustained—perhaps horseshoe-shaped—it is possible that corneal suturing might again give the patient a useful eye, when a pad and bandage application might result in adhesion of the iris with a gaping wound, necessitating removal of the eye. Such an accident as a

penetrating wound of the cornea may occur in a man's only good eye, and if this grafting can be done without the risk of the supervention of sympathetic trouble, it seems desirable to try it. A large corneal wound tends to gape, the anterior chamber does not reform, the iris becomes extensively adherent to the wound and there is the risk of infection.

DISCUSSION

MR. CYRIL WALKER: The eventual result of this fine and laborious work will be excellent. That was also the view expressed when Mr. Thomas read his more elaborate contribution in May.

MR. HUMPHRY NEAME: The section shown by Mr. Thomas, in which a portion of Descemet's membrane was reduplicated, is reminiscent of the sections seen in old interstitial keratitis. I have had such specimens to examine, either because of the supervention of glaucoma or because a blind eye had ruptured. Sections of such eyes show a very thick new formation of fibrous tissue behind the remains of the old cornea, and the obviously ruptured Descemet's membrane is curled up in the same manner and lies at the middle depth of the existing cornea. The new fibrous tissue is fixed and is most obvious in the lower part of the cornea, but in a very inflamed eye there is considerable formation of cellular material in the anterior chamber, which accumulates in the lower part.

MR. J. H. FISHER: I do not think that in the case described the endothelium has spread in from the periphery to cover the posterior surface of the graft and to secrete a new deposit of hyaline material. Apparently all the other tissues of the graft have survived well, and I see no reason why the endothelial cells, in such an experiment, should not also retain their vitality. It is not an ingrowing of epithelial cells from the periphery, but a survival of the cells on the deep surface of the graft, which, under the conditions of excessive activity, have produced a second layer of secretion.

MR. MAURICE H. WHITING: The value of the experiment lies in the possibility of usefully repairing wounds of the cornea. But when a cornea is wounded in actual life, the conditions differ from those in a laboratory experiment; the wound is caused by a relatively blunt instrument and so a good deal of injury is sustained by the cornea, and infection is likely to supervene. During the World War I tried on a number of occasions to preserve a wounded cornea by means of a flap, but the attempted suture under those conditions in order to secure the wound edges usually resulted in even greater damage; therefore, I used the simpler pad and bandage to keep the flap in position. I believe that conjunctival flaps are unsuitable under war conditions.

MR. F. JULER: Mr. Thomas seemed to use a suture for each lip of the wound.

COLONEL LISTER: I have seen two cases of corneal wound in which conjunctival flaps were successful.

MR. TUDOR THOMAS: I do not know whether the endothehlium survived in the cornea, but probably it did. I agree that wounds of the cornea in actual life are usually caused by a blunt instrument, and that

there is a liability to infection of the wound, but even so, for a plain incised wound suturing might be useful. In a clean wound, an edge-to-edge corneal suture is best. Animals tolerate corneal stitches very well, provided they are not inserted right through the cornea, but only half way through.

# Book Reviews

---

ANATOMICAL INVESTIGATION OF THE CORTICOFUGAL PATHS IN MONKEY AND RABBIT. DR. A. BIEMOND. Pp. 88, with 14 photomicrographs. Doctorate Thesis, Amsterdam, 1929.

During his systematic investigations of corticopetal optical conduction in Java monkeys in 1927, B. Brouwer questioned where the optical stimulations remain after reaching the area striata. In a series of monkeys and rabbits occipital and other cortical lesions were made and the secondary degenerations were studied in serial sections made by the Marchi method. Without doubt, important corticofugal connections were demonstrated with the corpus quadrigeminum as well as with the corpus geniculatum externum. Biemond systematically correlated this material for his doctorate thesis.

Ten Java monkeys' (*Cynomolgus fascicularis*) and four rabbits' brains were examined by the Marchi technic from sixteen to eighteen days after operation. The part of the brain that contained the corpus geniculatum externum, radiatio optica and area striata of the operated side was examined with controls from the crossed hemisphere. The frontal lobe, brain stem, medulla oblongata and spinal cord were also examined. Fibers were again found which had been separated from their cells of origin, and the direction of the conduction path was determined. Very fine amyelinated end-branches could not be traced. In determinating the degree of degeneration, quantitative differences between inner and outer parts in both sagittal strata were compared. The corpus suprageniculatum in the oval third of the corpus geniculatum externum could be seen only in some cases; it never showed signs of Marchi degeneration.

One-sided lesions on the area striata in two rabbits and seven monkeys after from twelve to eighteen days showed secondary degeneration in the homolateral stratum sagittale, the corpus quadrigeminum anticum and the corpus geniculatum externum, thus confirming the earlier observations of Probst for the cat and the dog and of Berl and K. H. Bouman for the rabbit. This demonstrates a corticofugal optical connection of the area striata with the corpus geniculatum externum. Distinct degeneration was also found toward the parietal gyri, while in the case of parietal lesions no sign of degeneration was seen in the occipital gyri.

In all cases of lesions of the area striata in the monkey the corticofugal degeneration in the corpus geniculatum externum was the most pronounced in that part where the macular field was located, according to Brouwer and Zeeman's investigations of the retinal projection. Experimentally, that part of the area striata was destroyed which may be considered to be the macular projection on the cortex according to van Heuven's observations. Lesions of large cortical areas outside of the area striata did not produce secondary degeneration in the corpus geniculatum externum. Not only is every retinal quadrant separately

projected on the cortex, but every retinal quadrant can be influenced from its own cortical projection area in a corticofugal direction. The same is true for the macular area.

Degeneration of the stratum medullare superficiale was found (confirming K. H. Bouman) in both monkeys and rabbits. A secondary degeneration was localized in the dorsal part of the brachium in monkeys with lesions of the area striata, in sharp contrast with the vertical position after destruction of parts of the retina. The corticofugal connection with the corpus quadrigeminum anticum probably passes through the pulvinar without giving fibers to this ganglion. The impression is given that the pulvinar in the monkey is not influenced along corticofugal paths.

The observations on the strata sagittalia confirm fully Flechsig's differentiation based on a myelogenetic basis. He localized the corticopetal system in the stratum sagittale externum and the corticofugal system in the stratum internum. The degeneration was found in the middle zone. This is the region where the corticopetal macular fibers pass. It seems that a similar orientation exists in the corticofugal system. Ophthalmologists may be interested in the following observations.

Striata lesions in the monkey produced degeneration in the fasciculus occipitoparietalis at the bottom of the parietal gyri. Its course is most probably through the deep hemispheric medulla. A direct connection with the temporal gyri could not be demonstrated. The degenerated callosal fibers that cross over into the other hemisphere did not extend into the occipital pole. This may be in favor of heterotopic callosal connections. Many fibers branched off and disappeared in the strata sagittalia. The existence of a heterotopic callosal connection between occipital and crossed temporal cortex seems most probable for the monkey. Parieto-occipital and temporal-pontine connections run separately to the corpus geniculatum, where they form a solid bundle at its medial side. This continues to the lateral part of the pes pedunculi and can be traced into the pons.

It is reasonable to accept the same corticofugal connection of the area striata with the corpus geniculatum externum for man as was found in the monkey. In both man and monkey the axis cylinders of the corpus geniculatum externum go exclusively toward the optical cortex. Lower optical reflexes, which progress subcortically, follow another path, chiefly via the corpus quadrigeminum anticum. An optical memory field, though it has not been localized, must exist. "Seeing" and "observing" may physiologically be explained as follows: The image is projected on the area striata and an association with like or similar memory images follows. If the similarity becomes identification, determined details must undergo a slight change in a positive or in a negative sense. This influence, either fortifying or inhibiting, is exercised via the motor cells in the deeper layers of the area striata on the corpus geniculatum externum (on each part of this separately). This refers only to unconscious observation. As soon as some association becomes connected with the seen image, it is plain that the incitation has extended along associative paths further into the cerebral cortex. This hypothesis explains two most important results. The corticofugal optical projection comes from the area striata and it shows an analogous localization, as does the corticopetal projection.

Thus a logical explanation for the so-called "hemianopic Aufmerksamkeitsschwäche" (observation weakness) is given. Perhaps the more sensitive corticofugal conduction has suffered, though the corticopetal conduction is uninfluenced. The often paradoxic discongruence existing between anatomic lesions of the area striata and the loss of function, as well as the not understood possibility of recovery from hemianopias caused by foci, may be better explained. The corticofugal conduction influences the image formation so that the loss of function due to a focus, which has not been completely destroyed, may vary. Regarding the possibility of restitution, a disturbance once present can slowly become compensated when the continuous corticofugal influence conduction of the corresponding geniculatum-part becomes installed on a higher plane. The phenomenon of sight and observation can be comprehended as a local, in essence subconscious, act, which cannot be considered as psychologic in the ordinary sense of the word.

Biemond presented his work under the title "Regarding the Third Optical Neuron" before the Amsterdam Neurological Society at its meeting of Nov. 7, 1929. It was published in the *Tijdschrift voor Geneeskunde,* April 5, 1930. The discussion was as follows:

Professor Winkler remains of the opinion that in addition to optical fibers, fibers from the lemniscus also penetrate the gelatinous substance of the corpus geniculatum laterale. Biemond had no proof of this.

Prof. K. H. Bouman thinks that one should be careful in assigning a function to the corticofugal path. He asks why the path from the optical cortex toward the roof of the corpus quadrigeminum anticum could not be the path along which "einstellungs" (fusion) reflexes of the eye take place. Connections of this body with the nuclei of the oculomotorius may sufficiently induce them. On account of time, Biemond did not go into this phase, which is treated in his thesis. There he refers to the third category of the reflexes of Munk, a category between the opticus reflexes and the "Sehreflexe," which latter Munk considered in connection with "einstell" (fusion) movements of the eye.

Professor Brouwer mentions that van Heuven believes that the development of the parietal lobes has gradually displaced the macular centers backward in man (see his Doctorate thesis). In the caudal part of field 17 of Brodman, all of the region is occupied by the macula, while the peripheral retinal regions are located forward. In this connection an observation by Prof. O. Foerster is of interest.

In a man in whom the occipital lobe was exposed, the occipital pole was excited with a weak faradic current. The patient saw a faint zone in the middle of his visual field. If Foerster stimulated more forward located parts of the area striata the patient saw something moving, first in the outer part of the visual field then slowly going toward the middle. Professor Foerster told the speaker that he had seen total restitution of hemianopia in a case in which the entire area striata was destroyed. Biemond believes that the clinical restitution possibility can better be understood if the corticofugal connections with the corpus geniculatum externum are accepted. The corticofugal conduction acts thus in conjunction with image formation and a disturbance from a focus, which if not fully destroyed may gradually become compensated for, when the continuous corticofugal influence of the corticopetal conduction of

the corresponding geniculatum can remain on a higher plane (niveau). This possibility is excluded by a complete destruction of the area striata, for then the corticofugal apparatus will also be entirely destroyed.

EDMOND E. BLAAUW.

MANUAL OF THE DISEASES OF THE EYE. BY DR. C. H. MAY, New York. Thirteenth edition. Price, $4. Pp. 461, with 374 illustrations. New York: William Wood & Company, 1930.

This new edition of the well known "May" has been carefully revised and some new matter added. A description of the slit lamp and corneal microscope is given by T. Harrison Butler. The author, with great industry, strives to keep this useful book up to date yet within bounds; that he succeeds is shown by the continued popularity it enjoys.

A. K.

# Directory of Ophthalmologic Societies *

## FOREIGN

### Oxford Ophthalmological Congress

Master: Dr. A. B. Cridland, Salisbury House, Chapel Ash, Wolverhampton, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.

## NATIONAL

### American Medical Association, Section on Ophthalmology

Chairman: Dr. George F. Suker, 25 E. Washington St., Chicago.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Philadelphia. Time: June 8-12, 1931.

### American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.

### American Ophthalmological Society

President: Dr. Arnold Knapp, 10 E. 54th St., New York.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.

## SECTIONAL

### New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Me.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

### Pacific Coast Oto-Ophthalmological Society

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

### Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

### Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Fred Hasty, 119 Seventh Ave., Nashville, Tenn.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Louisville, Ky. Time: November, 1930.

---

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

### Southwestern Michigan Triological Society

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

## STATE

### Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Donald H. O'Rourke, 217 Imperial Bldg., Denver.
Place: First floor, Metropolitan Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

### Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

### Eye, Ear, Nose and Throat Club of Georgia

President: W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts, Bldg., Atlanta.

### Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. A. Van Osdol, 320 N. Meridian St., Indianapolis.
Secretary: Dr. Robert J. Masters, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: December 10 and 11, 1930.

### Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. John C. Brown, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. J. H. Morse, 2511 Washburn Ave., S., Minneapolis.

### Montana Academy of Oto-Ophthalmology

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.

### North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

### Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. W. McCollom, 346½ Washington St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

### Rhode Island Ophthalmological and Otological Society

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

### Utah Ophthalmological Society

President: Dr. Franklin H. Raley, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. Leroy Smith, 1005 Medical Arts Bldg., Salt Lake City.

### Virginia Society of Oto-Laryngology and Ophthalmology

Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.
President: Dr. Fletcher D. Woodward, Box 162, University.

## LOCAL

### Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

### Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. Louis Joseph Sebert, 86 Bloor St., W., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

### Atlanta Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. McDougall, Atlantic National Bank Bldg., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

### Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Leo J. Goldbach, 1012 N. Charles St., Baltimore.
Secretary: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

### Brooklyn Ophthalmological Society

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

### Buffalo Ophthalmologic Club

President: Dr. A. F. Luhr, 40 W. North Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

### Chicago Ophthalmological Society

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday from October to May.

### Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

### Cleveland Ophthalmological Club

Chairman: Dr. W. E. Burner, Guardian Bldg., Cleveland.
Secretary: Dr. M. P. Motto, Rose Bldg., Cleveland.

### College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

### Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

### Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Decherd, 1717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

### Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

### Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

### Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 510 Main St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

### Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

### Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

### Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

### Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

### Los Angeles County Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. A. Ray Irvine, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month.

### Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

### Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

### Milwaukee Oto-Ophthalmic Society

President: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

### Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. R. J. Warner, 706 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

### New Orleans Ophthalmological and Otological Society

President: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

### New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. E. F. Krug, 12 W. 44th St., New York.
Secretary: Dr. William F. C. Steinbugler, 815 Park Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

### Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. George B. Potter, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 425 Aquila Court, Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

### Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

### Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

### Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. A. Vincent Walker, 833 South Ave., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

### St. Louis Ophthalmic Society

President: Dr. Charles W. Tooker, 308 N. 6th St., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

### San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. F. W. Sorrell, Gibbs Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. A. F. Clark, 728 Main Ave., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

### Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. A. N. Codd, 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Marc Anthony, Old National Bank Bldg., Spokane, Wash.

### San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Sigurd von Christierson, 11 W. Portal Ave., San Francisco.
Secretary: Dr. Wilber F. Swett, 693 Sutter St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

### Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.